VOLUME 51 NUMBER 3

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A HEARING DEFECT IS NOT A DISGRACE

There is no disgrace in being born deaf, no shame if hearing defects develop in later life. Yet many a family, ignorant of the cause of acoustical difficulties, suffer from "strong guilt feelings because the defect 'runs in the family.'" [1] With such a viewpoint it is natural to attempt to keep the defect secret. Lack of normal adjustment to the handicap is the inevitable consequence. This is especially true of the child, who acts as a barometer of his family's thinking.

Only when the causes of the loss of hearing are understood by everyone, only when the problem of a hearing defect is openly faced, only then will solutions be found.

CAUSES OF HEARING LOSS

Cause	Number
Focalized infections	
Mastoiditis	5
Ear abscess	5
Systemic diseases	
Scarlet fever	1
Colds	3
Measles	3
Chicken pox	1
Birth injury	3
Heredity	11
Accident	2
Cause unknown or not given	4
TOTAL CASES	38

HEARING DEFECTS ARE NOT NECESSARILY INHERITED

Perhaps hearing defects do "run in the family," yet the number of such instances is surely much less than the public believes. If both partners of a marriage are congenitally deaf, then there is an enhanced chance that the offspring will be similarly handicapped. However, when the hearing defects are not congenital, "the likelihood of deaf offspring is very slight." [2] Let us work to dispel the fear of deafness when no real reason for such fear exists.

THERE ARE MANY CAUSES OF HEARING DEFECTS

The causes of deafness are varied; heredity is but one of the less important. In the child, infections and their complications are those most commonly responsible for damage to the auditory apparatus. Here lies the greatest opportunity of the physician to prevent aural handicaps.

SOMETIMES HEARING DEFECTS CAN BE PREVENTED

Recently there seems apparent a small relative decrease in hearing defects.[2] This can largely be attributed to the newer methods of treatment of the infectious diseases of childhood.

But improved treatment of infectious diseases is not enough. Better, more thorough physical examinations in the school and in the physician's office will bring to light many conditions which need treatment, diseased tonsils, adenoids—among others—which may, if unrecognized or disregarded, lead to hearing defects in the young. The real tragedy of defective hearing in the child is that "so little is done about it by the parents," [3] that "as a rule the family physician is inadequately equipped to determine the true status of their condition." [3]

Consider only the fact that children with defective hearing lag three to four years behind normal children in their school [3] and it becomes apparent that the recognition, no less than the prevention of hearing losses, is of importance, beyond all estimation, to the child.

In the matter of preventing hearing loss in the adult, optimism is more difficult. "By what method can one avoid otosclerosis?" Furstenberg [4] enquired. By what method, indeed! And other causes of hearing loss in adults may be as difficult to handle. "Prevention," he wrote "is an admirable policy, . . . yet physicians must recognize its limitations. The point where prevention terminates and therapeutic procedures begin must be clearly discerned if practitioners are to achieve a solidarity of purpose and action that will function for the best interest of the person who is hard of hearing." Yet by routine aural examinations, by the prevention, if possible, of illnesses which might lead to further damage to hearing, much can be done to minimize the progress of the diseases which cause hearing defects.[5]

To prevent patients from suffering the confusion, the fatigue and depression of hearing defects, to bring those who have these difficulties back to normal social intercourse, by use of modern medical methods including scientific mechanical aids—these are mighty aims, but aims by no means beyond attainment.

"In recent years medical science has scored some great triumphs, and in the field of the prevention of deafness no little may be in store to be accomplished in the years to come." [6]

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5. Kinkade, J. M.: The Need of a Combined Program of Medical, Educational, Psychological and Vocational Rehabilitation of the Acoustically Impaired. In Aural Re-Education: Psychological and Therapeutic Aspects. Hearing Survey Quarterly, 1946, pp. 36-40.
6. Best, H.: Idem, Chap. 4.

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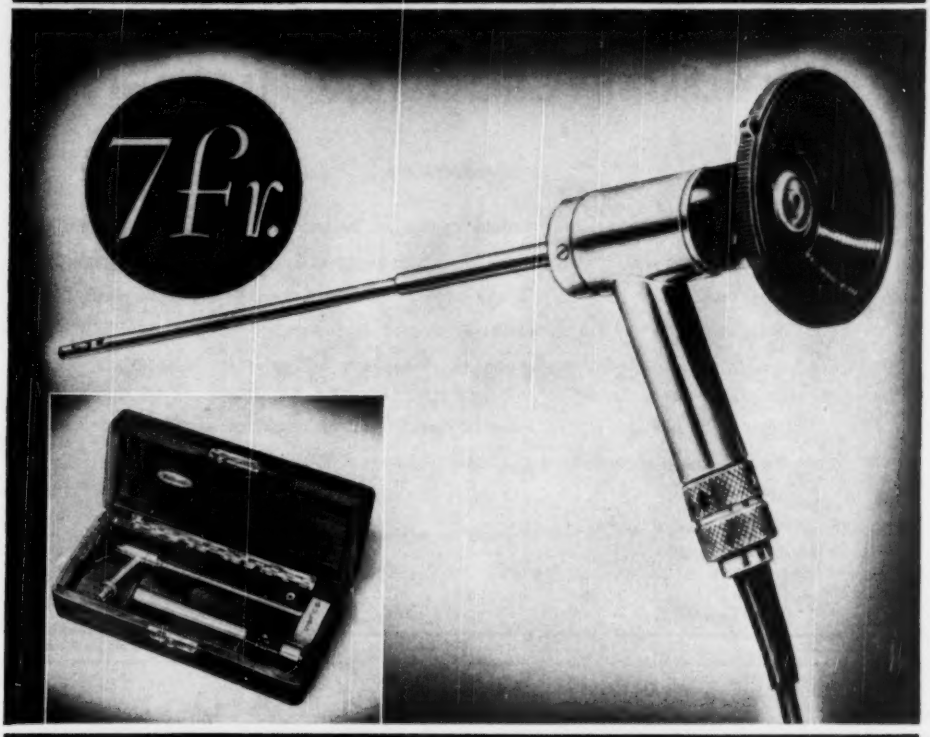

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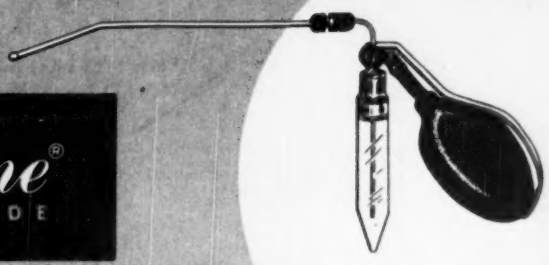

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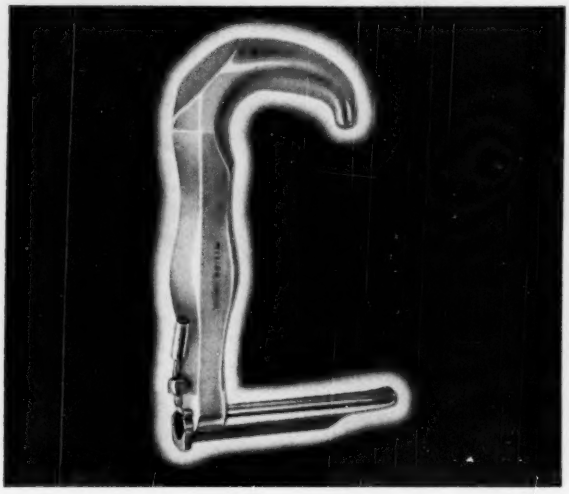

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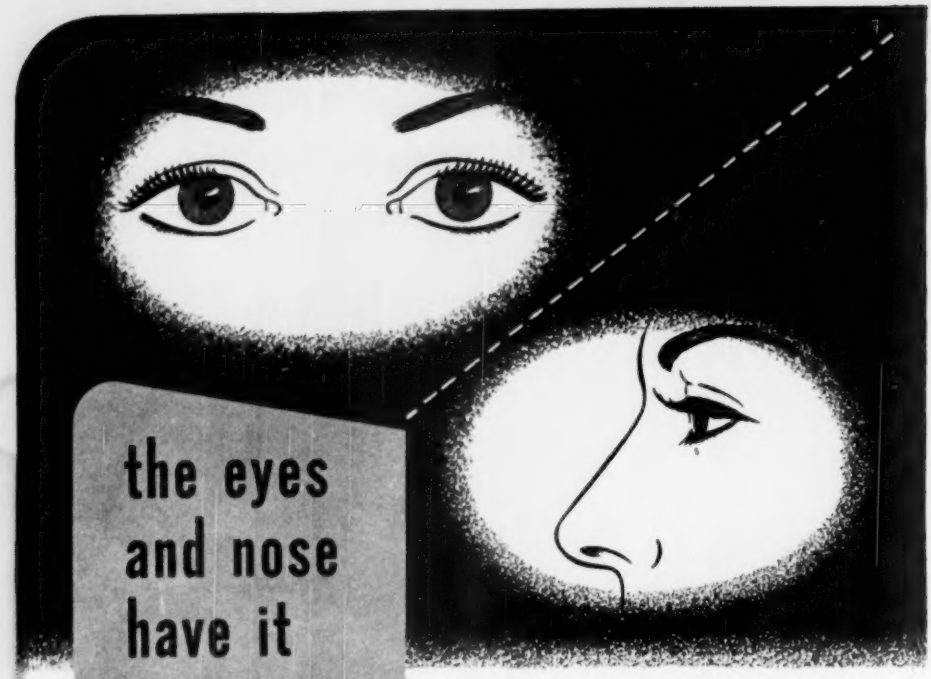

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1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

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THE COCHLEOGRAM AND ITS CLINICAL APPLICATION

Concluding Observations

JULIUS LEMPERT, M.D.
NEW YORK

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BOSTON

ERNEST GLEN WEVER, Ph.D.
AND
MERLE LAWRENCE, Ph.D.
PRINCETON, N. J.

IN AN ARTICLE which appeared in the ARCHIVES in January 1947,[1] we discussed in a preliminary way the possibility of making use of the electrical responses of the human cochlea in the diagnosis of diseases of the ear. This application obviously depends on the development of suitable technics for approaching the cochlea and applying an electrode in such a manner as to produce a faithful and reliable record of the electrical activity set up by the action of sounds. Our investigation of these technics has continued for more than three years, and we now are in a position to render a final report of the results of our efforts and to express our judgment regarding the practical value of the procedure.

In our exploration of this new method we have borne in mind its three special advantages for an indication of auditory function, advantages that have been fully revealed in the study of the ears of lower animals. In the first place, the method is wholly objective; it yields results that are precise and easily reproducible. In the second place, it reveals the response of the ear to any tone over the whole practical range of intensities and not merely at a threshold level. Finally, and most important of all, it represents directly the activity of the peripheral receptive mechanism without the complications introduced by the central nervous system. More specifically, it represents the activity of the organ of Corti, and therefore when we have the information that it yields we are able to make a clear distinction between sensory and neural deafness.

From the Lempert Research Foundation, Inc., Lempert Institute of Otology.

1. Lempert, J.; Wever, E. G., and Lawrence, M.: The Cochleogram and its Clinical Application: A Preliminary Report, Arch. Otolaryng. **45**:61-67 (Jan.) 1947.

Our experiments have taken the following course: We have first assured ourselves that the cochlear potentials are present in man as in other animals and have the same general character. Then we have confronted the technical problems of the most suitable form of electrode, its most favorable point of application and the means of making the application. In this investigation we have carried out tests on 32 human subjects (using one ear in each case) and on 16 monkeys (usually both ears).

NATURE OF THE POTENTIALS IN MAN

We have succeeded in observing the cochlear potentials in 13 out of the 32 human ears studied. In most of these the tests were made in the course of a fenestration operation, and in the others the tests were made during operations for the relief of tinnitus of Ménière's disease.

In most of the successful cases the responses were elicited by all sorts of aerial stimuli: voice sounds, various noises and pure tones from an audiometer. The responses were readily elicited also by bone conduction stimuli, produced by tuning forks applied to the bones of the skull. In a few ears in which the conduction deafness was particularly severe only the bone conduction tones were effective.

The responses were observed most readily to tones of the middle range, from 512 to 2048 cycles, and, occasionally, to lower and higher frequencies. The magnitudes of the potentials were small, of the order of a few microvolts.

The small magnitude of the potientials is to be attributed, in part, to the special conditions under which the experiments were carried out. Unavoidably, these ears were far from normal; they were seriously impaired in acuity by the disease processes that prompted the operation and were further modified by the operation itself. However, we are convinced that this is not the full explanation. The monkey ears, which were normal, gave potentials that likewise were smaller than those observed in the lower mammals. The maximum values obtained in the monkey are about 20 decibels below those obtained in guinea pigs and cats. It seems probable that there is some peculiarity of the primate ear that limits the available potentials.

This peculiarity might be an actual deficiency in the generation of electrical changes; yet our careful consideration of this possibility leads us to set it aside. The primate ear is quite as efficient in the medium high tone region as other mammalian ears in the reception and utilization of sounds. The primate ear possesses the same fundamental structures. The hair cells (where, we believe, the potentials are generated) are similarly situated so as to be acted on by acoustic vibrations, and they are present in even greater numbers; hence, we

infer that the generated potentials are at least as large in the monkey and man as in the other mammals.

We are led to the alternative conclusion that in the primate ear there are conditions that make it especially difficult to pick up the potentials with an electrode. A reasonable explanation rests on the peculiar bony enclosure of the cochlea. In the lower mammals the cochlea juts into the middle ear cavity and has a relatively thin bony capsule; in the primates, on the other hand, the cochlea is almost completely enclosed by thick, spongy bone. It may be that this bony enclosure provides good conductive paths about the cochlea and so effectively short circuits a large part of the sensory potentials.

TYPES OF ELECTRODE AND THEIR PLACEMENT

Numerous experiments on laboratory animals have shown that the most satisfactory electrode for general purposes is a small triangle of silver or platinum foil, about 0.0001 inch (0.003 mm.) in thickness, which is soldered to the end of a fine wire. The tip of the foil is applied gently to the surface of the round window membrane, to which it adheres. It is best that it should not touch the bony rim of the window, for any contact other than that with the membrane reduces the recorded potentials.

These animal experiments have shown further that an electrode on the bony capsule of the cochlea, in an ear such as the guinea pig's, in which the bony wall is thin, gives fairly satisfactory results also. This sort of contact is particularly favorable near the apical end of the cochlea where the wall is thinnest and when low tones are used as stimuli.

There is no doubt that this foil electrode would be the best for use in man also if it were possible to place it on the round window membrane. We have used it successfully in the monkey, though in this animal the anatomic conditions are far less favorable for approaching the round window than they are in most laboratory animals. There is no auditory bulla, the middle ear cavity is relatively small and the round window is partially obstructed by the tympanic membrane. With careful operative methods, however, an exposure can be made and the foil electrode applied. We have done this in 10 monkeys, often on both ears, and invariably have succeeded in observing the cochlear potentials.

In man, however, we are faced with greater difficulties. The anatomic conditions are even less favorable than in the monkey. The round window membrane is not easily approachable even when the middle ear cavity is widely exposed, for it lies concealed at the end of a relatively deep niche that is also somewhat curving. It is hardly

possible to insert the wire with its delicate foil tip so as to make contact with the round window membrane.

Our best success in man has been achieved with an electrode consisting of a moderately stiff wire of silver or copper whose end has been formed into a bead by heating it momentarily in a flame. The tip is bent to a suitable curve after the round window niche is exposed and its individual tortuosities are noted. Then, in most instances, it can be successfully inserted so as to bring the beaded end in the near vicinity of the round window membrane. Unfortunately, on account of the curved course of the niche, it is not usually possible to determine by direct vision whether the bead is resting on the membrane. To ascertain the location by tactual cues is difficult also, because oftentimes the niche contains a number of little nooks that catch and retain the bead thereby giving the impression that the end has been reached. Only in an exceptional ear is the round window niche shallow and straight enough to permit a simple, visually guided placement of the electrode. In these ears either the foil or the bead type of electrode may be used, and the two give the same results.

CLINICAL APPLICATION OF THE PROCEDURE

The procedures thus far described were carried out in the course of an operation on the ear and could be carried out only in this situation, when the ear was widely exposed to view. For general clinical applications, however, such an exposure is not available. We need to make our clinical diagnosis before major surgical intervention begins. This condition limits us to what may be done through the tympanic membrane without damage to it.

It is possible, as we have found, to pass a needle electrode through the tympanic membrane and to make contact with the bony promontory beyond. The needle electrode then may be maintained safely in position for the time necessary for a series of cochlear response tests. After the needle is withdrawn, the drum membrane heals perfectly in a short time.

Out of nine trials by this method we have obtained positive results in three. However, these results have been much inferior to the ones obtained with placements on the round window membrane. Only the strongest tones of the middle range were effective, and the potentials were small.

In this connection we have tried further measures. We have injected saline solution and other electrolytes through the drum membrane into the middle ear cavity, with the hope that their presence there might enhance the potentials recorded from the promontory. Unfortunately, these efforts have been unavailing. Uniformly, they have reduced rather than strengthened the observed responses. We

have been forced to conclude that in man, as is generally true in other animals, the bony capsule of the cochlea is not a very favorable location for the recording of the potentials.

At this point we turned our attention back to the round window. We conceived that if we could obtain a view of this window through the drum membrane it might be possible to apply an electrode to it. We sought the aid of Dr. Georg von Békésy, who provided an optical instrument for our purpose. The instrument is of the order of a cystoscope, but with two special features. The tube is particularly slender—1 mm. in outside diameter—and the illumination is external. The light from a small bulb passes through a lens system and is then reflected by a prism through the tube to another prism at its end. The terminal prism reflects the light at an angle to the object to be viewed and then returns the image of the object along the same path up the tube and straight on to the eye. To examine the middle ear structures a small slit is made in the drum membrane and the tube inserted. Because the field of view is very small, it is necessary to move the tube about and to build up a visualization of the structures. This is a difficult feat and becomes possible only after considerable practice. It is also hazardous, and great care must be used to prevent a laceration of the drum membrane. It is still not possible, however, to see into the round window niche, except in unusual instances in which this niche is shallow and laterally disposed. The anatomic situation in the large majority of ears and the risk of injury to the drum membrane discourage this approach toward a suitable placing of a recording electrode.

We have concluded, after these considerable efforts, that the recording of the cochlear potentials is not a practical clinical procedure. It can be done easily enough during a surgical operation, but then, of course, the clinical decisions have already been made. Without major surgical procedure—with only a drum incision—it is possible only in those rare instances in which the anatomic conditions are favorable, and then only with exercise of the utmost care and skill. A clinical method ought to be routinely applicable and reliable in results, and we have not been able to adapt the procedure to meet these conditions.

MÉNIÈRE'S SYNDROME

A New Drug for Control of the Acute Attack

MILES ATKINSON, M.D., F.R.C.S. (Eng.)
NEW YORK

THE COMPOUND 5-ethyl-5-(2-methylallyl)-2-thiobarbituric acid (called for experimental purposes V-12[1]) was prepared by Abbott Laboratories during the synthesis of a series of barbituric acid derivatives and was found to exhibit high efficiency as a motion sickness preventive (Noble[2]) in association with low hypnotic power. It seemed possible, therefore, that the drug might be effective in controlling the attacks of vertigo in cases of Ménière's syndrome, and an investigation was undertaken to determine this point.

MATERIAL AND PROCEDURE

Type of Case Investigated.—The acute attack in cases of Ménière's syndrome is of two types: (1) a sudden, severe attack of rotational vertigo, occurring without warning and followed, after a short interval, by vomiting; (2) a more gradual attack of less severity and longer duration, accompanied with nausea but seldom with actual vomiting, characterized by gross disturbance of balance without the sensation of rotation and often initiated by change of position—thus being more akin to motion sickness. In the rotational type of attack, medication orally is useless because the vomiting ejects the drug before it can take effect. The type of attack with which this report is concerned, therefore, is the positional only. Thirty-eight patients have been investigated with regard to control of such attacks with V-12. All these patients had been subject to such attacks for varying periods, were familiar with the prodrome, when present, of severe or mild attacks, and none had any means of controlling an attack once it had started.

Dosage.—V-12 was supplied for the investigation by Abbott Laboratories in two forms, alone and in combination with scopolamine hydrobromide U. S. P. (hyoscine hydrobromide). A capsule of the pure compound contained 200 mg.; a capsule of the combined drugs contained 150 mg. of V-12 and 0.3 mg. of scopolamine hydrobromide.

Administration.—The pure compound alone was used first. Patients were instructed to take 1 capsule as soon as they had warning of the onset of an attack. If no relief, or only partial relief, was obtained, a second capsule was to be taken after thirty minutes, and if relief was still incomplete, a third capsule was to be taken after another sixty minutes. Not more than 3 capsules were

1. V-12 is now marketed under the name of Mosidal® by Abbott Laboratories.

2. Noble, R. L.: Treatment of Experimental Motion Sickness in Humans, Canad. J. Research (Sect. E) **24**:10 (Feb.) 1946.

to be taken within two hours. If relief was still not obtained, the experiment was to be considered a failure.

RESULTS

V-12 Alone.—Thirty-two patients were treated. The effect of the drug was judged from the result obtained in at least two attacks, and many patients reported on more than this number. Twenty-one patients reported complete effectiveness, vertigo being consistently abolished, usually after 1 capsule, sometimes after 2 and rarely only after 3, the amount necessary depending on the severity of the attack: 6 patients obtained partial relief, a modification in the intensity of the vertigo to the extent of only a mild unsteadiness, whereas 5 reported no relief.

V-12 and Scopolamine Hydrobromide.—To the 11 patients who had obtained only partial or no relief, the combined capsule was given to be used in the same way as the capsule with V-12 alone. The 6 patients who had obtained partial relief with V-12 alone reported no apparent improvement in results with the combined capsule. Of the 5 patients who had obtained no relief from V-12 alone, 2 reported a somewhat questionable improvement with the combined capsule and 3 reported that relief was still not obtained. It appeared, therefore, that the addition of scopolamine hydrobromide in the dose of 0.3 mg. made no material difference to the effectiveness of V-12 for this condition. (Noble,[2] however, reported some increase in the effectiveness in motion sickness with the addition of scopolamine.)

V-12 and Riboflavin.—It has been reported elsewhere (Atkinson[3]) that the group of patients on whom this investigation has been conducted showed signs of riboflavin deficiency and that their tendency to recurrence of attacks could be controlled by the administration of riboflavin. In a few such cases it has been possible to control or modify an actual attack by intravenous injection of a large dose (20 to 30 mg.) of riboflavin. It seemed reasonable, therefore, to try the combination of riboflavin with V-12 by mouth. The 11 patients who had failed to obtain relief either with V-12 alone or in combination with scopolamine were instructed to take a V-12 capsule together with 50 mg. of riboflavin as soon after the onset of an attack as possible and repeat this combination as before, if necessary. Three of the 6 patients who had obtained partial relief reported complete relief with this regimen after only one dose, whereas previously three doses of V-12 alone had failed to produce more than partial relief; 2 of the other 3 patients

3. Atkinson, M.: Ménière's Syndrome: Observations on Vitamin Deficiency as the Causative Factor; I. The Vestibular Disturbance, Arch. Otolaryng. **49**:151 (Feb.) 1949.

reported much greater, though not total, relief in two severe attacks each and total relief in several moderately severe attacks. The sixth patient reported no change with the addition of riboflavin. Three of the 5 patients who had obtained no relief previously reported relief or decided improvement, depending on severity of attack, with the new regimen, whereas the other 2 reported no improvement. As controls, these 9 patients were subsequently given 50 mg. of riboflavin by mouth without V-12, which was without effect in sixteen attacks.

Thus, with the addition of riboflavin, 8 of the 11 patients who had obtained no relief with V-12 alone or in combination with scopolamine obtained total or considerable relief. In only 2 did the combination fail completely. In addition, 6 patients were treated with V-12 and 50 mg. of riboflavin without previous control with V-12 alone. All these patients reported relief.

TOXIC EFFECTS

Drowsiness was reported by 2 patients. In 1 patient it was of such mild degree that he continued to use the drug for the relief obtained from it. In 1 of the 2 patients with failure of results, the drowsiness was sufficiently marked on each of four attempts of the patient to take the drug that its use was discontinued. No other toxic effect was observed.

INTRAVENOUS ADMINISTRATION

A supply of the sodium salt of the drug for parenteral use was also made available and was used intravenously on 4 patients in six acute attacks accompanied with severe nausea or vomiting. One hundred and fifty milligrams of the drug was dissolved in 5 cc. of distilled water and injected slowly. In each instance narcosis was produced, lasting from five to fifteen minutes; on five occasions complete relief from vertigo was experienced on recovery, patients who had previously been completely incapacitated rising from the couch without qualm or discomfort. The remaining patient experienced pronounced though incomplete relief and was able to get up and go home immediately after recovery from the narcosis.

SUMMARY

V-12 alone produced complete relief in more than sixty attacks of positional vertigo in 21 of 32 patients with Ménière's syndrome.

The addition of scopolamine hydrobromide U. S. P. did not produce any apparent increase in effectiveness.

The addition of riboflavin produced definite improvement or total relief in 9 of 11 patients who had obtained only partial relief or no relief at all with V-12 alone. Six other patients treated by this regime from the beginning also obtained complete relief.

Drowsiness was noticed by 2 patients only; in 1 instance it was severe enough to cause discontinuance of the experiment.

Intravenous administration produced immediate complete relief from five acute attacks with nausea or vomiting in 3 patients and partial relief in the fourth. In each instance a short-lived but complete narcosis was produced.

CONCLUSION

V-12 is an effective drug for control of vertigo in that variety of Ménière's syndrome in which the onset of the attack is gradual. The effectiveness is increased when riboflavin is administered at the same time, an observation which makes it seem advisable to use this combination in all such cases.

36 East Thirty-Sixth Street.

OSTEOMA OF THE FRONTAL SINUS

Report of Five Cases

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OSTEOMA may be defined as a bony tumor, benign and discrete, with variations in the microscopic and morphologic appearance and of unknown cause. It may be considered malignant only in the sense that it has the capacity to grow, and in so doing to encroach on surrounding vital structures and menace the welfare and life of its host.

INCIDENCE

Osteoma of the frontal sinus is quite uncommon. I have discussed its occurrence with many otolaryngologists who are outstanding in their specialty and who have a very large practice, yet they have never seen a case of it. Most of the reports are of single cases, and the highest number reported by one man is 5, by Dowling[1] in 1945. In 1941 Teed,[2] in making a survey of all medical literature on the subject, found only 321 cases. In this survey he found a ratio of 172 men to 93 women. Statistics have shown that this lesion is commoner in youth and in men, as is well borne out in this series. However, one cannot consider this series as an index of the general populace, since the sex and age observed in an Army general hospital cannot be compared with that of a general civilian hospital. On the other hand, considering the large number of veterans and civilian dependents, both young and old, who are seen here daily, the difference cannot be too great.

ETIOLOGY

Although many theories have been postulated about the origin of osteoma, its specific cause is still unknown. Trauma has been stressed by some writers and, indeed, in my first case the patient was struck a heavy blow over the left supraorbital ridge and bridge of his nose by a Japanese soldier with the butt of a rifle two years prior to entry into this hospital. Shortly thereafter, vague, intermittent pain developed over this area, and the pain continued to increase up to the time of his admission

1. Dowling, J. R.: Osteoma of Frontal Sinus: Five Cases, Arch. Otolaryng. **41**:99 (Feb.) 1945.

2. Teed, W. R.: Primary Osteoma of the Frontal Sinus, Arch. Otolaryng. **33**:255 (Feb.) 1941.

to this hospital. It does not seem possible, however, that an osteoma could attain the size that this one did in two years. A few cases have been followed by otolaryngologists, who evidently did not believe in removal of this type of tumor until it was absolutely necessary. They found that the tumor was of extremely slow growth and that it required from three to five years after recognition to reach proportions great enough to erode the sinus wall.

Many writers believe that these osteomas arise from embryonic cartilaginous cells at the junction of the ethmoid and frontal bones; others believe that they originate from the periosteum of the sinus wall. However, the consensus at present is that osteomas develop from periosteal embryonal rests.

PATHOLOGY

Osteoma is a benign growth pathologically and rarely, if ever, recurs when completely removed. Ewing [3] stated that it may be ivory-like with solid lamellas and with few or no haversian canals, or it may be spongy with cancellous tissue and contain narrow cavities or spaces. Some osteomas, however, are of the mixed type, being composed of both eburnated dense bone and spongy bone. He further stated that it may be difficult to differentiate certain forms of osteoma from simple hypertrophy of bone. In the series presented here, all the osteomas were of the eburnated type.

SYMPTOMS AND DIAGNOSIS

The diagnosis of osteoma cannot be made in the absence of deformity without the use of the roentgenogram. In only 1 case in this series was there a deformity, and a tentative diagnosis was made prior to roentgenography. Many writers state that there are three cardinal symptoms: dizziness, headache and deformity. It is believed that dizziness is not a cardinal symptom; nor does it appear that this symptom could be produced unless the growth had reached a size large enough to exert pronounced pressure on the surrounding structures.

The symptoms produced by this tumor depend primarily on its location, the rapidity of growth and the direction of pressure. At times it may obstruct the nasofrontal duct and produce a mucocele, pyocele or symptoms of a vacuum type sinusitis. If the tumor extends into the orbit, ocular symptoms, such as diplopia, proptosis or exophthalmus, will be manifested, depending on the area of the orbit on which the pressure is produced. Erosion of the posterior frontal plate may result in meningitis, brain abscess or rhinorrhea. Involvement of the anterior frontal plate

3. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

will result in deformity, which may or may not produce symptoms and which may first be noticed by the patient himself or by the physician on examination.

One should not hesitate to order roentgenographic investigation when the entity is suspected, for roentgenograms not only confirm the diagnosis but outline the size of the osteoma and its proximity to vital surrounding structures.

PROGNOSIS

Since this tumor is benign and slow-growing, the prognosis is good. Beck [4] stated that without operation 48 per cent of the patients with osteoma will die of complications of the osteoma. Dowling [3] reported that since 1875 there have been 189 cases in which surgical treatment was undertaken, with a mortality of 3.7 per cent. The total mortality for the entire group of surgical cases is 7.76 per cent.[3]

Since the only cure for this condition is surgical removal, it would seem that the sooner the tumor is extirpated the less likely it is that complications would develop. Certainly, with surgery and anesthesia advanced to the state that it is today, the mortality rate would be extremely low. The sooner radical surgical extirpation is done after the diagnosis, the sooner one can hope for relief; the least deformity will be produced and the operative risk will be lessened. It is understandable, however, that if the tumor is discovered when it is small and asymptomatic and has not encroached on the opposite sinus wall, careful observation of its progress by repeated roentgenographic examinations may be justified. On the other hand, such examinations are expensive and lead to apprehension and anxiety on the part of the patient.

Before surgical treatment is instituted in this hospital, the patient is thoroughly examined and medical and laboratory studies are made. The patient is placed on a schedule of treatment with penicillin, 50,000 units every three hours, twenty-four hours prior to operation and administration is continued for four days thereafter.

The high brow incision has been used exclusively and has been found to leave a minimal scar and to afford adequate exposure in all cases in which the tumor has not extended to the superior portion of a large or high sinus. If extension has occurred, the incision should be made higher up on the forehead in a horizontal plane and should follow a normal crevice of the skin. After removal of the osteoma, its pedicle is curetted thoroughly, sulfanilamide crystals are dusted into the sinus cavity and the incision closed around a small rubber catheter placed at its lateral margin. The nasofrontal duct is not probed.

4. Beck, J. C.: Some Uncommon Types of Neoplasms About the Head, Tr. Am. Laryng., Rhin. & Otol. Soc. **36**:374, 1930; Laryngoscope **40**:511 (July) 1930.

Forty-eight hours after operation, the sinus is irrigated with warm isotonic sodium chloride solution through the catheter, all excess fluid removed by gentle suction and the catheter then removed.

REPORT OF CASES

Seven cases of osteoma were seen in this hospital within a period of one year, 5 of which are herewith reported. In 2 cases in which the osteoma was small and asymptomatic operation was not performed, and they were therefore not included in this series.

In the presentation of the following cases, all laboratory data and physical findings were within normal limits and accordingly will not be included.

CASE 1.—A man aged 36 was first seen at the ear, nose and throat clinic in an Army general hospital in November 1946, with a history of acute coryza of three days' duration. Physical examination at that time indicated nothing essentially abnormal, with the exception of numerous pedunculated polypi in the left nasal passage, which produced approximately 90 per cent obstruction. No pus was noted. On transillumination, the left frontal sinus was dark. On palpation, a definite deformity of the medial third of the left supraorbital ridge was noted. This deformity appeared to be due to erosion of the orbital ridge, with replacement by a hard bony mass, which was nontender, immobile and protruded approximately 0.5 cm. beyond the orbital line into the orbit.

Roentgenograms of the sinuses revealed a dense bony mass, completely filling the left frontal sinus; the mass had eroded a portion of the medial third of the supraorbital ridge.

On being further questioned, the patient stated that he had been struck over this area with the butt of a rifle by a Japanese soldier two years before his admission to the hospital, and since that time he had noticed mild pain over the area. The pain was localized, was intermittent and had increased in severity, but it was not severe enough for him to seek medical aid. Two weeks after his admission to the hospital, and after the coryza had subsided, a polypectomy was performed. He was discharged four days later and readmitted on Feb. 1, 1947. On February 12, with the patient under general anesthesia, the osteoma was removed. At the time of operation it was noted that the bony tumor had eroded an area approximately 1.5 cm. in diameter of the anterior wall of the sinus and supraorbital ridge. The tumor was attached by a pedicle in the nasofrontal duct. The mass was removed intact and measured 3.6 by 2.2 by 1.8 cm.

The pathologic report follows.

Gross Examination.—The specimen consisted of a dome-shaped elevation of hard bony tissue, approximately 0.5 cm. above the surface and 0.4 cm. in diameter. The base of this mass was 0.2 cm. in thickness and 1.0 cm. in diameter. The tissue was dense, solid and cancellous.

Microscopic Examination.—The section consisted of decalcified bone, which was seen as a pinkish, homogeneous-staining substance, through which could be seen linear-like striations. This material was densely vascular, and the vascular channels were congested, showing no evidence of fibrosis or cellular infiltration. The bone could be seen in areas to be laid down in concentric-like rings, with central areas of lacunas as well as haversian canals. The diagnosis was osteoma of frontal sinus. This report represents the picture described by the pathologist in all 5 cases.

The patient made an uneventful recovery and was discharged from the hospital on the tenth postoperative day. He was seen eighteen months later, at which time he was completely free from symptoms. Surprisingly enough, there was no deformity. Roentgenograms at this time showed no recurrence of the tumor, and the frontal sinus was aerated.

CASE 2.—A white man aged 32, was first seen in this clinic on Feb. 26, 1947. He had been a patient in this hospital for the previous eighteen months, undergoing reconstructive surgery of the left femur because of a compound, comminuted fracture incurred during World War II.

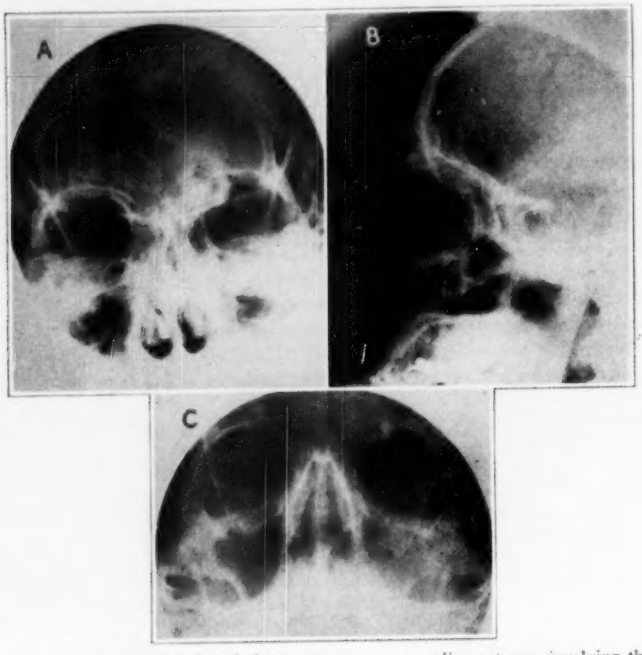

Fig. 1 (case 1).—*A*, *B* and *C*, roentgenograms revealing osteoma involving the entire left frontal sinus, eroding the supraorbital ridge and extending into the orbit.

During this time he complained of headache in the right frontal region of three years' duration. The headache was first dull, throbbing and intermittent, but later it became continuous. He stated that during the previous six months it had become progressively worse, with such increasing frequency of exacerbations that it was unbearable. The pain would often radiate to the occiput, becoming severe in the occipital region. At first he was given acetylsalicylic acid and later codeine, with some relief from symptoms, but recently nothing had given relief.

Physical examination, including the cranial nerves, indicated nothing abnormal. Roentgenograms of the cranium and sinuses were requested only as a routine procedure. The following report was received:

"There is a small osteoma in the right frontal sinus, which is quite irregular in shape and extends from the posterior to the anterior walls of the sinus."

Ten days later, after clearance from the orthopedic section, the patient was operated on with general anesthesia. The tumor was found to have eroded through the anterior wall of the sinus and periosteum, producing a porous or sievelike appearance; the periosteum was so closely adhered to the wall that it could not be removed. The pedicle was attached to the posterior wall a few millimeters from the septum. The osteoma measured 2 by 1 by 1 cm.

This patient made an uneventful postoperative recovery and was seen frequently in the hospital over a twelve month period. The headaches had completely disappeared; however, he did have a slight postoperative depression over the right frontal area, because the high location of the tumor necessitated a high incision.

This case proved to be such an interesting one that the patient was presented to the tumor board and staff of this hospital. During the presentation, it was brought out that two different consultations with the neuropsychiatric service had been obtained prior to the ear, nose and

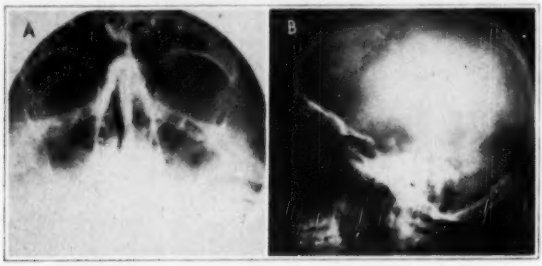

Fig. 2 (case 2).—*A* and *B*, roentgenograms revealing a small, irregular osteoma in the superior medial portion of the right frontal sinus.

throat consultation, because his symptoms were believed to be on a psychoneurotic basis.

Case 3.—A white man aged 34, was referred to this hospital on May 9, 1947, with a diagnosis of "neoplasm of the right frontal sinus." He stated that one month previously a peculiar sensation developed over his right eye, which he described as numbness rather than actual pain. At times he had noticed a feeling of tenderness on pressure over and under the supraorbital ridge, which, he believed, had increased in severity. He reported to his local hospital, where roentgenograms that were made revealed an irregular, opaque mass in the right frontal sinus.

Five days later, with the patient under general anesthesia, the osteoma, which was attached to the septum at its inferior portion by a hard, bony pedicle, was removed. It measured 2 by 1.5 cm. The patient made an uneventful recovery; when he was seen six months later, he was completely free from symptoms. There was no postoperative deformity.

Case 4.—A white man aged 33, was transferred to this hospital on Aug. 18, 1949, with a diagnosis of "frontal sinus tumor, cause undetermined." He presented a history of headaches in the frontal region of two years' duration, which he described as being localized in the midfrontal area and at times radiating posteriorly, causing a moderately severe pain at the base of the skull. The frontal pain

was dull and persistent, and rarely was the patient completely free from symptoms. The severity of the symptoms had not increased during the past two years. The patient had no other associated symptoms, and results of the physical examination was negative throughout.

Roentgenograms of the sinuses revealed an osteoma in the right frontal sinus. The osteoma was rectangular in outline in the anteroposterior view and appeared to approximate closely the medial sinus wall.

One week later, with the patient under general anesthesia, the osteoma was removed through a high brow incision. The growth had eroded the anterior wall

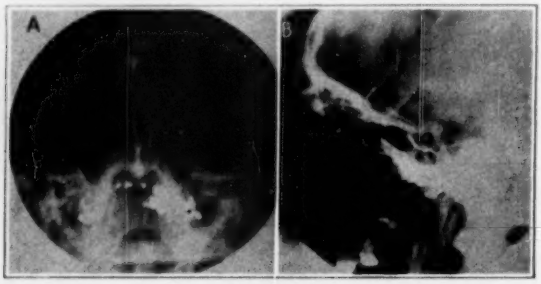

Fig. 3 (case 3).—*A* and *B*, roentgenograms revealing an irregular, lobulated osteoma of moderate size in the right frontal sinus.

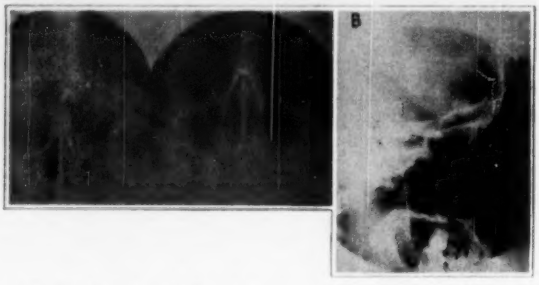

Fig. 4 (case 4).—*A* and *B*, roentgenograms revealing a small rectangular-shaped osteoma in the right frontal sinus.

of the frontal sinus, as in case 2, and was attached rather firmly to the intersinal septum; this necessitated entering the left frontal sinus. No pedicle could be demonstrated, for the osteoma was firmly attached to the septum and the anterior wall of frontal sinus.

The patient made an uneventful postoperative recovery, and on discharge ten days later he was completely free from symptoms. There was no postoperative deformity.

Case 5.—A white obese woman aged 39 entered this clinic on Oct. 6, 1947, complaining of attacks of severe headache in the frontal region of three years' duration. The pain originated high in the midfrontal area and radiated downward,

under the left eye. The patient described the pain as dull, throbbing and continuous. There were frequent exacerbations of sharp, severe pain, which radiated posteriorly and produced soreness and stiffness of the muscles of the neck. These severe attacks necessitated her seeing the family physician, who prescribed "a nasal spray and narcotics," from which she obtained temporary relief.

The personal and family history revealed that the patient's husband had died approximately one year prior to the onset of her symptoms and left her with two small children and a son in the Army, who was her only support. The allotment she received was not adequate to support the family, so that it was necessary for her to seek common labor employment. It was at this time that the first symptoms appeared. With this in mind, it was difficult not to feel that there was some psychogenic factor contributing to the symptoms and, perhaps, causing the greater part of them. However, roentgenograms of the sinuses showed a small osteoma in the floor of the left frontal sinus. This tumor aroused a great deal of interest and speculation about whether it was strictly in the frontal sinus or in the lateral

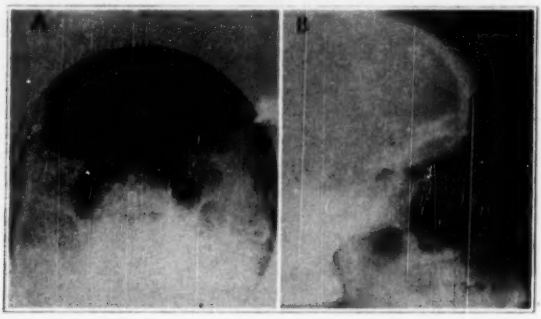

Fig. 5 (case 5).—*A* and *B*, roentgenograms revealing a small osteoma in the floor of the left frontal sinus.

portion of the ethmoid sinus. With this finding, the staff was of the opinion that the patient should have the benefit of surgical procedure. She was admitted to the hospital and operated on one week later. The osteoma was approached through a midbrow incision and was found to be attached by a medium-sized bony pedicle, just posterolateral to the nasofrontal duct, over a moderate-sized ethmoid cell, which was broken into during the procedure. The tumor on removal measured 1.0 by 0.5 cm. in diameter.

The patient made an uneventful recovery and left the hospital on the sixth postoperative day. She was observed in the clinic over a four month period, during which time she remained completely free from symptoms. During this time she had remarried. It was only through my own professional curiosity that I had her return at two week intervals, and after a four month period I was satisfied that the symptoms had been real. As a consequence, I told her not to return. To my surprise, she returned two weeks later, complaining of the same type of pain she had presented originally. Roentgenograms revealed no recurrence. A placebo was prescribed, which gave her complete relief. She was last seen one year after the operation, at which time she had no complaints.

SUMMARY

Primary osteoma of the frontal sinus is considered an uncommon lesion.

Five cases are presented in which the ages ranged between 32 and 39 years. One patient was a woman.

Three patients presented symptoms of a similar pattern, e. g., frontal pain which radiated to the occiput, producing soreness and stiffness of the posterior cervical muscles. This mechanism of referred pain has not been clearly explained.

There are no cardinal symptoms. The roentgenogram is the only means of making a definite diagnosis.

The treatment is surgical.

CONCLUSIONS

1. An osteoma of the frontal sinus, although small, may produce definite and sometimes severe pain, which, in all probability, is due to the involvement of the overlying periosteum.

2. Osteomas producing symptoms should be removed, regardless of their size.

3. Erosion of the sinus wall by the osteoma in the absence of symptoms should be considered an absolute indication for surgical intervention.

SURGICAL DECOMPRESSION OF MALIGNANT EXOPHTHALMOS

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MALIGNANT exophthalmos [1] is a clinical entity distinct from exophthalmos as generally seen accompanying thyrotoxicosis. These conditions may be closely related etiologically [2]; yet the diverse clinical pictures they present and the difference in their management make it necessary that the clinician be acquainted with the diagnostic criteria of the disease and the surgeon be familiar with the underlying pathologic process on which the treatment is based.

In this paper I shall enlarge on these facts and introduce a new surgical approach, which I have employed successfully.

HISTORY

Malignant exophthalmos was first seen and described by von Basedow [3] in 1840. Since this initial report, 216 cases have been reported in the literature. (Würdemann and Becker,[4] Lahey,[5] Moore,[6] Zimmerman,[7] Hirsch and Urbanek,[8] Naffziger and Jones,[9] Gasteiger,[10]

1. Known also as hyperophthalmopathic Graves's disease, malignant exophthalmos or thyrotropic exophthalmos. Graves's disease is classic exophthalmic goiter, thyrotoxic exophthalmos or toxic diffuse goiter.

2. Means, J. H.: Hyperophthalmopathic Graves' Disease, Ann. Int. Med. **23**:779-789, 1945.

3. von Basedow, C.: Exophthalmos durch Hypertrophie des Zellgewebes in der Augenhöhle, Wchnschr. f. d. ges. Heilk. **6**:197-204 and 220-228, 1840.

4. Würdemann, H. V., and Becker, W.: Atypical Exophthalmic Goiter with Endothelioma of the Pituitary and Thyroid Bodies: Death from General Sepsis: Autopsy, Ophthalmology **2**:411-427, 1905-1906.

5. Lahey, F. H.: Loss of Both Eyes from the Exophthalmos of Hyperthyroidism, Boston, M. J. **182**:427, 1920.

6. Moore, R.: Exophthalmos and Limitation of the Eye Movements of Graves' Disease, Lancet **2**:701, 1920.

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(Footnotes continued on next page)

King,[11] Semmes,[12] Andersson,[13] Merrill and Oaks,[14] Bothman,[15] Means,[16] Hertz,[17] Thomas and Woods,[18] Rosenbaum,[19] Brain,[20] Stallard,[21] Lyle,[22] Kistner,[23] Ginsburg,[24] McCravey and Mather,[25] Reichling and Marx,[26] Meesmann,[27] Poppen,[28] Mulvany,[29] Bardram,[30] Zondek

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11. King, B. T.: The Course of Exophthalmos, West. J. Surg. **39**:602-609, 1931.

12. Semmes, R. E., in discussion on papers of Dinsmore, Naffziger and Jones, and Clute and Veal, J. A. M. A. **99**:645-646 (Aug. 20) 1932.

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14. Merrill, H. G., and Oaks, L. W.: Extreme Bilateral Exophthalmos: Report of Two Cases with Autopsy Findings in One, Am. J. Ophth. **16**:231-236, 1933.

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16. (*a*) Means, J. H.: The Nature of Graves' Disease, with Special Reference to Its Ophthalmic Component, Am. J. M. Sc. **207**:1-19, 1944; (b) personal communication to the author; (*c*) footnote 2.

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18. Thomas, H. M., Jr., and Woods, A. C.: Progressive Exophthalmos Following Thyroidectomy, Bull. Johns Hopkins Hosp. **59**:99-113, 1936.

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20. Brain, W. R.: Exophthalmic Ophthalmoplegia, Quart. J. Med. **7**:293-323, 1938.

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23. Kistner, F. B.: Decompression for Exophthalmos: Report of Three Cases, J. A. M. A. **112**:37-38 (Jan. 7) 1939.

24. Ginsburg, S.: Postoperative Progressive Exophthalmos with Low Basal Metabolic Rate, Ann. Int. Med. **13**:424-450, 1939.

25. McCravey, A., and Mather, H. R., Jr.: Treatment of Exophthalmos by Decompression of the Orbit: Report of Two Cases, Arch. Neurol. & Pyschiat. **43**:187-189 (Jan.) 1940.

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28. Poppen, J. L.: Exophthalmos, Am. J. Surg. **64**:74-79, 1944.

29. Mulvany, J. A.: The Exophthalmos of Hyperthyroidism: Differentiation in Mechanism, Pathology, Symptomatology and Treatment of Two Varieties, Am. J. Ophth. **27**:589-612, 693-712 and 820-832, 1944.

30. Bardram, M. T.: Progressive Exophthalmos, Acta ophth. **22**:1-26, 1944.

and Ticho[31] and Mann.[32] In all probability, however, many more patients than those reported on have been seen by clinicians and surgeons, since 102 patients were observed and treated by three workers alone.[33]

Nevertheless, the disease is much rarer than exophthalmos as seen in classic toxic diffuse goiter, of which it is one of the cardinal symptoms.

INCIDENCE

Age and Sex.—Malignant exophthalmos is seen three to four times as often in men of the older age groups as of the younger.[29] In a large series of Osler, exophthalmos in classic toxic diffuse goiter was found to be eight times as common in women as in men.[34] Most of these patients belonged to the younger groups between the ages of 20 and 40.

Geography.—Most of the cases reported have been from England and the United States. One is inclined to think, therefore, that these countries have a greater incidence of malignant exophthalmos than continental Europe.

CLINICAL FEATURES

Symptoms and Signs.—As a rule, the disease has an insidious onset. The ocular changes may or may not be accompanied with systemic evidence of toxic diffuse goiter. Most frequently, cases of this type have been described as following thyroidectomy performed either for thyrotoxicosis with and without exophthalmos or for what appeared to be classic exophthalmos without toxic manifestations but which was exceedingly aggravated by the operation. In most spontaneous cases, the development of symptoms usually requires three to four months.[20] Cases developing after thyroidectomy have a latent period from as short as ten days to as much as two years after the operation.[18] The patient complains of protrusion of the eyeballs, noticed either by himself or by his friends. There is pronounced lacrimation, photophobia, difficulty in convergence, blurring of vision and diplopia. There is decided swelling of the lids, and closure of the eyes is difficult, this situation resulting in an uncomfortable and dangerous dryness of the cornea.

On physical examination, extreme bilateral exophthalmos with chemosis and tearing are the cardinal findings. The degree of pro-

31. Zondek, H. and Ticho, A.: Observation on So-Called Thyrotropic Exophthalmos, Brit. M. J. **1**:836-838, 1945.

32. Mann, I.: Exophthalmic Ophthalmoplegia and Its Relation to Thyrotoxicosis, Am. J. Ophth. **29**:654-673, 1946.

33. (*a*) Means.[16b] (*b*) Mulvany.[29] (*c*) Naffziger, H. C.: Progressive Exophthalmos Associated with Disorders of the Thyroid Gland, Am. Surg. **105**:529-544, 1938.

34. Osler, W.: Modern Medicine, revised and re-edited by T. McCrae, ed. 3, Philadelphia, Lea & Febiger, 1927, vol. 5, p. 355.

trusion of the two eyes may vary. Attempts at reduction cause severe pain to the patient and impart a ligneous sensation to the examining finger. A widened palpebral fissure frames a bulging, irritated conjunctiva, which prolapses from the conjunctival sac. Active and even passive closure of the lid is almost impossible. The cornea appears dull, and in advanced cases ulcerations may occur, leading to perforations and eventually to ophthalmia. Horizontal and vertical motion of the eyeball is impaired in the majority of patients. Upward movement is disturbed earliest and most frequently; lateral movement is affected less, and depression least of all.[29]

Differential Diagnosis.—The symptomatology and physical findings of malignant exophthalmos are so strikingly different from those of

Fig. 1.—Malignant exophthalmos (case of Dr. S. L. Gargill, Beth Israel Hospital). Notice the widened palpebral fissure, framing the bulging conjunctiva, which prolapses from the conjunctival sac.

classic toxic diffuse goiter that little difficulty should be encountered in differentiating the two, especially in advanced cases. Classic exophthalmos is almost always accompanied with systemic evidence of thyrotoxicosis. As a rule, the patient does not experience any subjective ocular difficulties unless an accidental injury or infection occurs. There is no edema of the lids, chemosis, eversion of the conjunctival sac or limitation of motion or vision, but rare instances of paralysis of the lateral rectus and ptosis have been observed, resulting in weakness of lateral gaze and diplopia. There is no lacrimation. Palpation reveals an elastic eyeball, and digital pressure does not result in pain; the bulbus can be displaced posteriorly. Thyroidectomy occasionally improves the condition but does not aggravate it. Local surgical intervention is not indicated.

A variety of other conditions may result in bilateral exophthalmos, such as thrombosis of a cavernous sinus, cerebral tumors, hydrocephalus, oxycephalia, osteitis deformans and Hand-Schüller-Christian disease. These can be differentiated on the basis of collateral findings.[35]

PATHOGENESIS AND PATHOLOGY

Two approaches to the etiology of this disease have been made. It has been considered a phase of toxic diffuse goiter by one group of investigators,[36] who therefore called it hyperophthalmopathic Graves's disease. Those of another school of thought [29] contended that the clinical manifestations and underlying endocrinologic disorder are sufficiently different to make malignant exophthalmos a disease entity the pathogenesis of which differs from that of thyrotoxicosis. Mulvany found support for his viewpoint in experiments with animals treated with thyroid-stimulating hormone. Though there seems to be a wide gap between the opinions of these two groups of investigators, both groups agreed on the probable influence of the thyroid-stimulating hormone of the pituitary gland in the causation of malignant exophthalmos.

Autopsies have been performed in cases of thyrotoxicosis and malignant exophthalmos. The gross and microscopic findings differ decidedly. Hypertrophy of the external ocular muscles equivalent to an increase of three to eight times normal is the major pathologic change in malignant exophthalmos.[37] Microscopically, one notices this increase in volume to be due to edema and proliferation. Round cell infiltrations and fragmentation of muscle fibers are constant features.

The morphology of the orbital contents in classic exophthalmos of toxic diffuse goiter is in keeping with the systemic myasthenia commonly found in this disorder.[38] The muscles are atrophic, with increased fatty deposits and pronounced increase of the total fat content of the retrobulbar tissue.[39] The histologic appearance of the external ocular muscles shows scattered wasting of muscle fibers, loss of striation and fibrillations. The size of the muscles is not strikingly altered.

35. It may be of interest that von Basedow,[3] in his original publication, set out by eliminating the possibility of exophthalmos being due to paralysis of the external ocular muscles, osteomalacia, periostitis, exostosis of the orbit, polypoid diseases of the sinuses, tumors of the brain, dermoid of the orbit, scirrhus of the lacrimal gland, traumatic blood effusions, cellulitis of the orbit and hydrophthalmos.

36. Hertz.[17] Means.[16a,b]

37. Moore.[6] Naffziger and Jones.[9]

38. Askanazy, M.: Pathologisch-anatomische Beiträge zur Kenntniss des Morbus Basedowii, insbesondere über die dabei auftretende Muskelerkrankung, Deutsches Arch. F. klin. Med. **61**:118-186, 1898.

39. Rundle, F. F., and Pochin, E. E.: The Orbital Tissues in Thyrotoxicosis: A Quantitative Analysis Relating to Exophthalmos, Clin. Sc. **5**:51-74, 1944.

Malignant exophthalmos can be explained on a purely mechanical basis due to increased muscular bulk.[40] The mode of production of proptosis, as seen in simple exophthalmos of thyrotoxicosis, however, is still debated. Loss of extraocular muscle tonus resulting in imbalance between striped and unstriped orbital musculature, together with increased orbital fat, are held responsible for part of the changes.[29]

MEDICAL TREATMENT

Treatment is medical or surgical, depending on the degree of exophthalmos and on the degree to which the cornea is endangered. It has already been emphasized that 50 per cent of the cases of malignant exophthalmos developed after thyroidectomy and that the condition is made worse by the thyroidectomy.[17] So treatment for this condition must avoid not only surgical but also so-called medical thyroidectomy (use of thiouracil).[41]

Medical therapy can be used in cases in which the exophthalmos is not too excessive and the cornea not endangered. Iodine is sometimes helpful [42] but is much more effective if it is given in conjunction with thyroid.[17] The dose of thyroid administered in these cases is 5 grains (0.3 Gm.) per day, but it can be increased to 7½ and to 10 grains (0.5 to 0.6 Gm.), depending on the patient's tolerance.[29] The rationale of this treatment is the inhibition by the thyroid hormone of the thyroid-stimulating hormone of the anterior lobe of the pituitary gland. It is best to reduce the dose of thyroid as the patient improves. Instead of iodine, Mulvany [29] advocated a daily dose of 5 to 10 mg. of diethylstilbestrol U. S. P., together with thyroid, especially for postmenopausal women.

Roentgen ray treatment, directed to the orbit, the thyroid and the pituitary gland has been given a trial. Irradiation of the orbital contents is of little or no value. It has been contended that irradiation of the thyroid gland enhances the effect of the iodine-thyroid regimen.[17] Irradiation of the pituitary gland has been recommended only recently by Ginsburg,[24] Means [2] and Mulvany [29]; each one has reported 1 case with successful treatment. Mulvany also reported a case with no benefit from his management. He stated that the condition in its early stages may be more amenable to this type of treatment.

OPERATIVE TREATMENT

Operation on the Lids and Orbital Content.—Tarsorrhaphy is a palliative measure which does not bring permanent relief but considerably diminishes the visual fields and gives a poor cosmetic result.

40. Naffziger, H. C.: Pathologic Changes in the Orbit in Progressive Exophthalmos, Arch. Ophth. **9**:1-12 (Jan.) 1933. Moore.[6]

41. Hertz,[17] Means [2]; footnote 16*b*. Mulvany.[29]

42. von Basedow,[3] Hertz.[17]

Lid suturing is performed routinely by Poppen [28] before surgical decompression.

Removal of Orbital Fat by Intraorbital Approach (Moore's Approach,[6] 1920).—Moore was the first to call attention to the feasibility of relieving pressure within the orbit by diminishing its contents. He removed orbital fat through the orbit by making an incision through the entire length of the inferior conjunctival fornix. He then exposed the contents of the orbit and picked away piecemeal with forceps as much orbital fat as possible. Altogether, a heaping teaspoonful was removed. Afterward, the lids were easily approximated and were in part sewn together over the cornea. At this occasion he exposed the extraocular muscles and found them to be greatly swollen, fusiform bellies instead of thin, flat and ribbon-like structures.

Operation on the Orbital Walls (Decompression Procedures).—(I shall discuss the various procedures in the chronologic order of their development.)

Removal of the Orbital Floor (Hirsch's Approach,[8] 1929): In 1929 Hirsch operated on a man aged 24 who had suffered from proptosis for five months. A thyroidectomy had been performed three months after the onset of his complaints. The exophthalmos did not improve after the operation; in fact, the patient thought that the proptosis had become more pronounced. Because of the increasing severity of the disability, a tarsorrhaphy was performed on the left eye. It was believed that a similar operation on the right eye would limit the visual fields too much, and, consequently, a decompression procedure by way of the maxillary sinus was decided on.

With the use of local anesthesia, the right maxillary sinus was entered from the canine fossa and the orbital floor exposed in its entirety. A part of the orbital wall of the maxillary sinus was chiseled away on both sides of the infraorbital nerve. The remaining orbital floor was removed with bone forceps, leaving only a narrow bony bridge for the infraorbital nerve, to avoid anesthesia of the cheek, and a bony support for the eyeball. The periorbital fascia was incised and the orbital fat then prolapsed into the maxillary sinus. Part of the fat was removed. The operation was terminated with establishment of suitable drainage in the inferior meatus, and the incision was then sutured. Success of the operation was clearly visible on the third postoperative day. Congestion of the scleral and conjunctival vessels had decreased and the eyeball could be displaced posteriorly somewhat; displacement had been impossible preoperatively. The patient was able to close his lids actively, and during sleep the cornea was covered by the upper lid, removing the danger of keratitis.

The exophthalmometric (Hertl) reading was 27 mm. before operation and 24 mm. two weeks after operation. The therapeutic result was highly satisfactory.

The degree of decompression can be increased by removal of the bony bridge containing the infraorbital nerve,[43] but loss of sensation or hypesthesia of the cheek will ensue. The decompression was performed through the orbital floor of the maxillary sinus in order to protect the orbital contents from nasal infection and emphysema of the orbit, which could be a real danger if the decompression were performed by removal of the lamina papyracea and the ethmoid cells. The creation of a wide opening for drainage minimizes the possibility of infection.

Meesmann [27] followed this method in 1939, but apparently he removed the whole floor of the orbit together with the infraorbital nerve. The exophthalmometric reading showed a recession of 7 mm. A corneal ulcer which had been present before operation healed.

Later methods relieved the pressure by removal of the superior, lateral or medial walls.

Removal of the Orbital Roof (Naffziger's Approach,[44] 1931): Naffziger reflected bilateral frontal flaps, elevated the underlying dura of the frontal lobe, unroofed the orbits and then removed the upper portion of the optic foramen. Preliminary roentgenographic studies are necessary to ascertain the extent to which the orbital plate is formed by the ethmoid and frontal sinuses. If a large enough portion of the orbital roof is removed the eyeball will recede decidedly, but the cerebral vascular pulsations are transmitted to the eyeball more or less noticeably.

Naffziger [28a] used this method in 31 cases and Poppen [28] in 28. Other cases of operation by this method have been reported on by Dandy,[45] Sachs,[46] Cairns,[47] Semmes,[12] Rosenbaum,[19] Kistner,[28] and McCravey and Mather.[29] Naffziger reported four failures and two deaths. Poppen mentioned two failures. Because postoperative corneal ulcers developed in 4 of his patients, Poppen supplemented the procedure by routine tarsorrhaphy. Three of his patients had transient, pronounced symptoms referable to the frontal lobe.

43. Even after surgical removal of the maxilla, this ligament (named Lockwood's ligament) keeps the globe in an approximately normal position (Kronfeld, P. C.; McHugh, G., and Polyak, S.: The Human Eye in Anatomical Transparencies, Rochester, N. Y., Bausch & Lomb Press, 1943, p. 34-35).

44. Naffziger, H. C.: Progressive Exophthalmos Following Thyroidectomy: Its Pathology and Treatment, Ann. Surg. **94**:582-586, 1931.

45. Dandy, W., cited by Thomas and Woods.[18]

46. Sachs, E., cited by Merrill and Oaks.[14]

47. Cairns, H., cited by Brain.[20]

Removal of the Lateral Wall (Swift's Approach,[48] 1935): The lateral orbital wall and arch are exposed and removed through a skin incision. The defect is covered by the orbital periosteum and the temporal muscle. McCravey and Mather[23] operated on 1 patient, using Naffziger's method on one side and Swift's on the other. The results were equally good, but Swift's method was considered less formidable. Thomas and Woods[18] found that the procedure gave good results in their hands, whereas Andersson[13] found it only partially satisfactory.

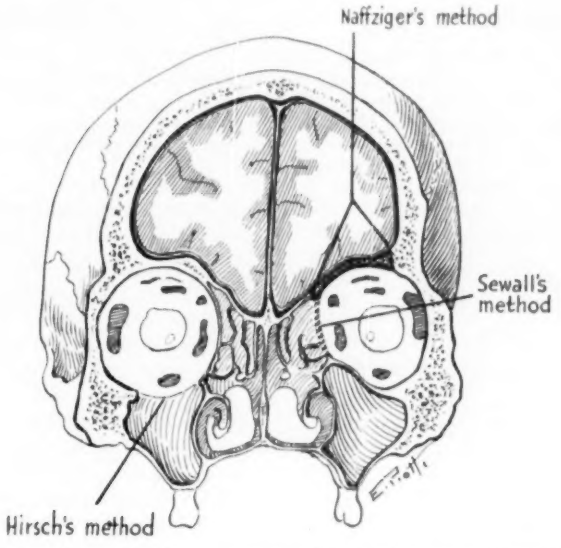

Fig. 2.—Diagram showing the methods of Hirsch, Naffziger and Sewall in the surgical treatment of malignant exophthalmos.

Removal of the Medial Orbital Wall (Sewall's Approach,[49] 1936): Sewall incised the skin of the orbit on its medial aspect then removed the lamina papyracea. When the ethmoid sinus extends into the orbital roof, its medial part may be removed too.

The method which was worked out in the laboratory was successfully applied by Kistner[23] and Schall and Reagan,[50] who each performed

48. Swift, G. W.: Operative Approach for Exophthalmos and Operative Approach, West. J. Surg. **43**:119-126, 1935.

49. Sewall, E. C.: Operative Control of Progressive Exophthalmos, Arch. Otolaryng. **24**:621-624 (Nov.) 1936.

50. Schall, L. A., and Reagan, D. J.: Malignant Exophthalmos, Ann. Otol., Rhin. & Laryng. **54**:37-42, 1945.

two operations. In 1 case postoperative meningitis developed, which was cured by sulfonamide therapy. The eyeball receded from 3 to 5 mm.

Evaluation of Methods.—The comparative advantages and disadvantages of the methods are tabulated.

Tarsorrhaphy	Palliative only
	Visual fields markedly diminished
	Cosmetically unsatisfactory
	Advisable only in mild cases
Moore	Removal of orbital fat by intraorbital approach
	Recession of minor degree followed by tarsorrhaphy
Hirsch	Simple procedure
	No external scar
	Minimal danger of infection
	Large potential space for decompression
	Recession from 3 to 7 mm.
Naffziger	Recession varying from minor to extreme degree
	Visualization and widening of optic foramen possible, if necessary
	Major procedure, leaving external scar
	Occasional fatality
	Limitation of the procedure, depending on the anatomy of the orbital roof
	Postoperative pulsation of the eyeball
	Temporary symptoms referable to the frontal lobe
	Indication in failure of a less risky operation and with papilledema
Swift	Satisfactory recession possible
	Limited space for decompression
	Major operation
	External scar
	Lack of protection from injury to the orbit
Sewall	Sufficient space for decompression
	Exposure of orbital contents to nasal infection
	External scar
	Recession of 3 to 5 mm.
	Risk of emphysema

SUMMARY

The diagnosis and pathogenesis of malignant exophthalmos is discussed.

The medical and surgical treatment of the disease is described.

A new method of Hirsch is described, in which the orbit is decompressed by removal of the orbital floor via the maxillary sinus.

The relative advantages and disadvantages of the various surgical methods are evaluated.

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ROLE OF STREPTOMYCIN IN LARYNGEAL TUBERCULOSIS

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FOR THE past two years, in our services at the Philadelphia General, the Episcopal, the Pennsylvania and the Abington Memorial hospital, we have been observing a group of patients with laryngeal tuberculosis. These patients, 37 in all, were given 0.5 Gm. of streptomycin intramuscularly, twice a day, during periods varying from two weeks to six months, for an average of three months per patient.

The dose of 1 Gm. per day was used because all the available literature indicates that this amount can be used safely over a long period. The patients were studied by means of frequent clinical observations, caloric tests and whispered voice tests after which, audiograms were made when considered necessary and a series of moving pictures of the larynx taken in color by one of us (W. A. L.).

In a review of the literature [1] on the use of streptomycin for pulmonary tuberculosis, it was mentioned that in a total of 223 cases in seven Army, Navy and Veterans Administration hospitals little or no improvement was shown in the fibrocavernous or proliferative cases, whereas in 85 per cent of the cases with exudative lesions there was some improvement. The dose was 1.8 Gm. per day for one hundred and twenty days (much larger than the dose in our cases of laryngeal tuberculosis). Tinnitus was present in 46 per cent of the cases, vertigo in 92 per cent, and hearing decreased in 2 per cent of the cases. Deafness seemed reversible one month after use of the drug was discontinued. Among our cases, we found that deafness was not reversible in 1 case of diminished hearing, and six months have already elapsed. Of the 233 cases, there was diminished or no response to caloric stimulation in 77 per cent.

Of 34 cases of tuberculous laryngitis at Olive View Sanatorium, there was recovery in 13 and improvement in 17; in 4 the condition

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1. The Effect of Streptomycin upon Pulmonary Tuberculosis: Preliminary Report of a Cooperative Study of Two Hundred and Thirty-Three Patients, Streptomycin Committee, Veterans Administration, Am. Rev. Tuberc. **56**:485 (Dec.) 1947.

became worse. Black and Bogen [2] concluded from this study that 2 Gm. doses were too large and 0.5 Gm. too small, but that doses of 1 Gm. were just about right.

Brown and Hinshaw,[3] of the Mayo Clinic, in reporting on 23 cases of tuberculosis of all types, with reference to the influence of streptomycin on the eighth nerve, stated that there was no true objective or subjective vertigo present; when the patients [2] held their feet together and their eyes closed, some became giddy, some looked to the wall for support and 1 had spontaneous nystagmus. There was no loss of hearing when the patients were getting only 1 to 2 Gm. per day. Brown and Hinshaw stated that the lesion was peripheral and not central. (No one has yet proved this, and it is open to considerable conjecture.) Also they were of the opinion that if streptomycin was discontinued at the onset of tinnitus, the discontinuation would probably prevent serious deafness.

The Council on Pharmacy and Chemistry of the American Medical Association [4] found streptomycin of value against gram-negative bacillary infections; however, it was found to be quite toxic for the eighth nerve and the kidneys.

Feldman and Hinshaw [5] found that guinea pigs could be inoculated with tuberculosis bacilli experimentally, and then after the disease was present for six weeks it could be suppressed with fairly large doses of streptomycin.

Feldman, Hinshaw and Karlson [6] used 64 guinea pigs in another study and found that it was not necessary to administer streptomycin at frequent intervals and that good results could be obtained by administering it twice a day.

Figi, Hinshaw and Feldman [7] reported 1 case of papillary tuberculous laryngitis in which 0.8 to 1.0 Gm. of streptomycin was given every twenty-four hours parenterally (in eight equal doses) for six weeks, and by aerosol, 0.5 Gm. in 30 cc. of isotonic sodium chloride solution, given in equal doses at the beginning of each hour for ten hours each

2. Black, M., and Bogen, E.: Streptomycin in Tuberculous Laryngitis, Am. Rev. Tuberc. **56**:405 (Nov.) 1947.

3. Brown, H. A., and Hinshaw, H. C.: Toxic Reaction of Streptomycin on the Eighth Nerve Apparatus, Proc. Staff Meet., Mayo Clin. **21**:347 (Sept.) 1946.

4. Streptomycin, Report of the Council on Pharmacy and Chemistry, J. A. M. A. **135**:839 (Nov.) 1947.

5. Feldman, W. H., and Hinshaw, H. C.: Chemotherapeutic Testing in Experimental Tuberculosis, Am. Rev. Tuberc. **51**:582 (June) 1945.

6. Feldman, W. H.: Hinshaw, H. C., and Karlson, A. G.: Frequency of Administration of Streptomycin, Am. Rev. Tuberc. **55**:433 (May) 1947.

7. Figi, F. A.; Hinshaw, H. C., and Feldman, W. H.: Treatment of Tuberculosis of the Larynx with Streptomycin: Report of a Case, Proc. Staff Meet., Mayo Clin. **21**:127 (March) 1946.

day. However, the patient had no real thoracic lesion but did have sputum positive for tubercle bacilli. One year later the patient felt good and the larynx was normal. But since there was no involvement of the chest at all, one can hardly draw any conclusions from 1 simple, uninvolved case.

An interesting study was made of 13 patients by Steenken, D'Esopo and Wolinsky [8] concerning the actual amount of streptomycin found in certain body cavities. With doses varying between 1.8 and 2 Gm. per day, it was found that the average concentration in large tuberculous cavities, pleural spaces and the tracheobronchial tree is much greater than concentrations which inhibit growth in vitro. It would have been even more noteworthy if a smaller dose of 1 Gm. per day, such as we used, had been employed.

Withers,[9] in a more recent study of tuberculous laryngitis, reported 12 cases, with recovery in 10 and improvement in 2. There were no changes whatsoever in the vestibular mechanism and few or no other toxic symptoms.

PRESENT STUDY

In the accompanying tables are a complete list of the patients, an itemized account of each patient, with a description of the larynx before and after treatment with streptomycin, the exact dose given, the general state of health and the responses to the drug, locally and systemically, and, finally, an over-all appraisal of the results obtained.

ANALYSIS OF DATA

We noticed that most of our patients had serious pulmonary tuberculosis of the fibrocavernous type, bilateral in scope, complicating the picture of extensive laryngeal involvement. There were some very speedy responses to the drug. Most of the patients had relief from pain and dysphagia very quickly, often in a matter of three or four days; there was a feeling of well being never experienced before or to such an extent with the usual treatments—voice rest, heliotherapy, use of chaulmoogra oil, roentgen ray therapy, injection of the superior laryngeal nerve, section of the superior laryngeal nerve and cautery.

There was considerable gain of weight in the large majority of cases, pronounced relief from hoarseness, and, in the cases of early involvement, including moderate edema, inflammation and infiltration of the larynx, there was almost a complete reversal to normal. Of course this was not

8. Steenken, W., Jr.; D'Esopo, N. D., and Wolinsky, E.: Excretion of Streptomycin in Tuberculous Cavities, the Pleural Space and the Tracheobronchial Tree, Am. Rev. Tuberc. **56**:403 (Nov.) 1947.

9. Withers, B. T.: Streptomycin in Treatment of Laryngeal Tuberculosis, Ann. Otol., Rhin., & Laryng. **57**:769 (Sept.) 1948.

TABLE 1.—*Summary of Data on 22 Patients with Laryngeal Tuberculosis Treated with Streptomycin*

Case	Age, Yr.	Race	Sex	Condition of Larynx: Before Streptomycin	Condition of Larynx: After Streptomycin	Dosage	General Health After Treatment	Labyrinthine Involvement
1	35	N	F	Cords and arytenoids edematous and inflamed	Much improved; only slight reddening of arytenoids	1 Gm. daily for 4 mo.	Much improved; hoarseness disappeared; 14 lb. weight gain	Total absence of vestibular responses; dizziness from 3rd day of therapy; just recovering from vertigo 1 mo. after treatment stopped; falls to the left; Romberg's sign present
2	24	N	M	Moderate involvement; cords and arytenoids swollen, infiltrated and reddened	Much improved; returned almost to normal	1 Gm. daily for 3 mo.	Patient felt fine; appetite improved; 12 lb. weight gain	Normal labyrinth; Romberg's sign not present; no tinnitus
3	57	N	F	True and false cords and epiglottis moderately involved	Much improved; disappearance of swelling and redness	1 Gm. daily for 4 mo.	Much improved; hoarseness and dysphagia disappeared; 22 lb. weight gain	Diminished responses; no reaction on right side to caloric tests; slight vertigo for 1 day
4	21	N	F	Mild involvement; some swelling, redness and edema of cords and arytenoids	Returned to normal appearance	1 Gm. daily for 9 wk.; some aerosol treatment for 42 days; 1.5 cc. twice a day (1 cc. contains 10 mg.)	Noticeably improved; hoarseness disappeared; 22 lb. weight gain in 5 mo.	Normal responses; Romberg's sign not present; no vertigo; no tinnitus
5	48	..	M	Moderate involvement of true and false cords; swollen and edematous arytenoids	Improved; arytenoids still reddened	1 Gm. daily for 6 wk. (perhaps should have been more)	Some improvement; some hoarseness still present; 14 lb. weight gain during treatment, followed by 6 lb. loss later	Diminished responses; part of vertical canal did not respond to caloric test; Romberg's sign not present; no vertigo
6	39	N	M	Moderate involvement; cords and arytenoids reddened and edematous; increase in interarytenoid space	Cords and arytenoids improved	1 Gm. daily for 5 wk. (should have been more)	Improved; much less hoarseness; better appetite; 7 lb. weight gain in 4 wk.	Normal responses; Romberg's sign not present; no vertigo or tinnitus
7	..	N	M	Pronounced involvement of true and false cords, arytenoids and epiglottis	Larynx improved for first 3 mo. of treatment; relapse to former condition occurred and continued in spite of 2d course of therapy	1 Gm. daily for two 3 mo. courses, 6 mo. apart	Hoarseness disappeared at first, then returned and is now persistent; patient failed to gain weight permanently	Patient always felt dizzy while taking streptomycin; free from vertigo 1 mo. after treatment; diminished responses; no response to caloric test for vertical canals on right; little response for horizontal canals on right; Romberg's sign not present
8	33	N	F	Pronounced involvement of entire larynx; ulceration, edema and inflammation of epiglottis, arytenoids and cords	Moderate improvement; less edema and redness, although much of the pathologic conditions still remained	1 Gm. daily for 2½ mo.	Patient died of advanced pulmonary and laryngeal tuberculosis; pain and dysphagia were relieved at first; later a severe relapse with return of all symptoms occurred	Normal responses; Romberg's sign not present
9	43	W	M	Pronounced ulceration, infiltration, edema and inflammation of entire larynx	Remained the same; decided involvement	1 Gm. daily for 24 days	Dermatitis, erythema multiforme (necessary to stop streptomycin); patient died from toxic erythema and advanced pulmonary tuberculosis	Severe illness prevented tests
10	32	N	..	Considerable involvement and ulceration of true and false cords; arytenoids reddened and edematous	Ulceration healed; cords and arytenoids much improved	1 Gm. daily for 3 mo.	Improved; 10 lb. weight gain; hoarseness and dysphagia disappeared	Diminished responses; no reaction on the left to caloric test; Romberg's sign not present

TABLE 1.—*Summary of Data on 22 Patients with Laryngeal Tuberculosis Treated with Streptomycin*

Case	Age, Yr.	Race	Sex	Condition of Larynx: Before Streptomycin	Condition of Larynx: After Streptomycin	Dosage	General Health After Treatment	Labyrinthine Involvement
11	45	W	M	Pronounced involvement of cords and arytenoids (inflamed and edematous); epiglottis involved	Improved; less edema and inflammation of entire larynx	1 Gm. daily for 3 mo.	Improved; 12 lb. weight gain; hoarseness and dysphagia disappeared	Normal responses; Romberg's sign not present
12	47	W	M	Considerable involvement of epiglottis, cords and arytenoids (edema, inflammation and ulceration)	Improved; less edema and inflammation of entire larynx	1 Gm. daily for 7 wk.	No weight gain; dysphagia and hoarseness relieved; (patient should have had further therapy	Normal responses; Romberg's sign not present
13	41	N	M	Moderate edema and inflammation of cords; retropharyngeal abscess incised	Much improved; returned to normal	1 Gm. daily for 3 mo.	Improved; 8 lb. weight gain; throat normal	Normal responses; Romberg's sign not present
14	38	N	M	Marked involvement of cords and arytenoids (ulcerated and of moth eaten appearance)	Improved moderately; less swelling of cords and arytenoids; ulceration cleared	1 Gm. daily for 30 days (not long enough)	Relapse occurred; hoarseness and dysphagia returned after initial disappearance; patient died of advanced pulmonary and laryngeal tuberculosis	Normal responses
15	38	W	F	True and false cords and arytenoids thickened and inflamed	Only slightly improved; true and false cords still showed thickening	1 Gm. daily for 7 wk.	No weight gain; but patient felt somewhat better	Dizziness about 4th weeks of therapy; no dizziness 3 wk. after therapy; Romberg's sign not present; normal response to labyrinth tests
16	27	N	F	Cords and arytenoids reddened and infiltrated	Much improved; cords and arytenoids almost normal	1 Gm. daily for 6 mo.	19 lb. weight gain; patient felt better	Normal labyrinth; no dizziness; Romberg's sign not present
17	46	N	M	True and false cords and arytenoids reddened and thickened	No improvement; patient received streptomycin for only 15 days	1 Gm. daily for 15 days	No weight gain; patient did not feel better; (streptomycin stopped because of deafness)	Normal labyrinth; Romberg's sign not present; deafness and tinnitus within 2 wk. of therapy; deafness has remained
18	60	N	F	Cords and arytenoids reddened and thickened	Much improved; almost normal	1 Gm. daily for 6 wk.	24 lb. weight gain; patient felt better; less coughing and hoarseness	Diminished response; no reaction of vertical canals on left side to caloric tests; Romberg's sign not present
19	37	..	F	Cords and arytenoids reddened and inflamed	Unimproved	1 Gm. daily for 2 mo.	No weight gain; gradual lowering in general appearance	Diminished response; vertigo from streptomycin; no reaction on right side to caloric tests; no reaction of vertical canals on left to caloric test
20	47	..	M	Pronounced edema of epiglottis; cords and arytenoids thickened and reddened	Epiglottis, cords and arytenoids improved	1 Gm. daily for 3 mo.	Weight gain; patient felt better within 1 wk.	Diminished responses; no reaction on right side to caloric tests; Romberg's sign not present
21	45	W	M	Epiglottis and aryepiglottic folds edematous; arytenoids infiltrated	Some improvement; less edema of epiglottis; cords and arytenoids slightly improved	1 Gm. daily for 2 mo.	Somewhat improved; patient felt better; sore throat disappeared; condition of lung worse; patient signed out against advice; tuberculous meningitis appeared to be developing	Normal responses
22	..	W	F	Epiglottis, cords and arytenoids edematous and inflamed	Much improved; epiglottis, cords and arytenoids noticeably reduced in size	1 Gm. daily for 3 mo.	Weight gain; patient felt better at once; (needed thoracoplasty but refused)	Normal response; Romberg's sign not present

TABLE 2.—*Summary of Data on Remaining Patients*

Case	Age, Yr.	Race	Sex	Condition of Larynx: Before Streptomycin	Condition of Larynx: After Streptomycin	Dosage	Results	8th Nerve Complications	Comment
23	18	W	F	Granuloma on right side	Reduced to ½ normal size	0.5 Gm. twice a day for 27 days	Very good for duration of treatment	Severe dizziness	Dysphagia improved
24	52	W	M	Diffuse ulceration of entire larynx	Mucous membrane smooth; no ulceration	0.5 Gm. twice a day for 76 days	Remarkable improvement; weight gain	None	No dysphagia
25	66	W	F	Ulceration of epiglottis	Ulcerations improved	0.5 Gm. twice a day for 56 days	Local discomfort disappeared; general improvement	None	No dysphagia
26	52	W	F	Ulcer on brim of epiglottis	Cleared	0.5 Gm. twice a day for 62 days	Very good	None	Patient is well
27	68	..	M	Ragged mucous membrane on right side of larynx	Ulcerations disappeared	0.5 Gm. twice a day for 45 days	Very good	None	Patient is well
28	62	W	M	Crater ulcer on left ventricle	No improvement	0.5 Gm. twice a day for 32 days	No change	None	Biopsy indicated carcinoma
29	55	W	M	Ulceration of right side of epiglottis	Only slight thickening of epiglottis	0.5 Gm. twice a day for 62 days	Definite improvement	None	No local discomfort
30	47	W	M	Ulceration of entire left side of larynx	Ulceration disappeared; thickening still present	0.5 Gm. twice a day for 45 days	Good	None	Dysphagia relieved
31	46	W	F	Hypertrophy and erosion of both cords	Mucous membrane smooth	0.5 Gm. twice a day for 36 days	Good	None	No local pain
32	51	W	F	Pronounced ulceration of mucous membrane of posterior commissure	Ulcerations disappeared	0.5 Gm. twice a day for 78 days	Good	Slight dizziness	Pronounced improvement
33	45	W	F	Knoblike projection of posterior commissure; erosion of mucous membrane	No change	0.5 Gm. twice a day for 48 days	No improvement	None	Projection removed by direct laryngoscopy
34	26	N	M	Pronounced ulceration	Moderate improvement	0.5 Gm. twice a day for 60 days	Fair improvement	Slight	Dysphagia improved
35	24	N	F	Larynx filled with polypoid tissue	No improvement	0.5 Gm. twice a day for 36 days	No change	None	Polyps of larynx
36	42	W	M	Erosion of all mucosa	Erosion disappeared	0.5 Gm. twice a day for 56 days	Weight gain	None	Pronounced improvement
37	38	W	M	Erosion of margin of both cords	Cleared	0.5 Gm. twice a day for 46 days	Good	Slight dizziness	No pain

true in the cases in which there were extensive ulceration, fibrosis and perichondritis, which had distorted the entire larynx before treatment was started.

Summary of Results.—Of 37 patients with laryngeal tuberculosis who were treated with 1 Gm. of streptomycin per day for approximately three months, we found that 21 improved considerably; 8 improved moderately; 5 remained unimproved or had relapse, and 3 died from severe bilateral fibrocavernous pulmonary tuberculosis. The toxic reactions to the streptomycin included diminished vestibular responses (9 patients); total abscence of vestibular responses (2 patients); severe dermatitis within twenty-four days (1 patient); diminished hearing within fifteen days, which has remained after six months, (1 patient)

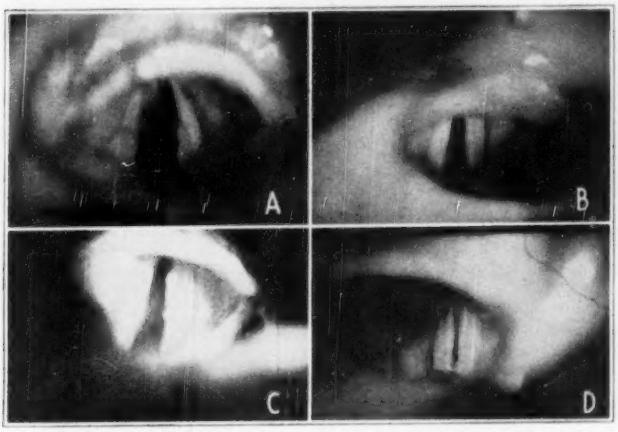

Fig. 1.—Photographs (case 11) taken before (*A*) and after (*B*) daily administration of 1 Gm. of streptomycin for ninety days; photographs (case 30) taken before (*C*) and after (*D*) daily administration of 1 Gm. of streptomycin for forty-five days.

and vertigo and tinnitus (1 patient). For this patient streptomycin had to be discontinued after twenty-seven days, but she improved with streptomycin in spite of the short duration it was used.

Comment.—In this series of 37 patients treated with streptomycin, all who had tuberculous involvement of the larynx showed a definite response. One patient (case 33) who had a small tuberculoma on the posterior margin of the right vocal cord, in addition to a generalized involvement of the mucous membrane, did show improvement in the appearance of the mucous membrane; but the tuberculoma persisted and had to be removed by direct laryngoscopy. After removal of the tuberculoma, the larynx cleared completely.

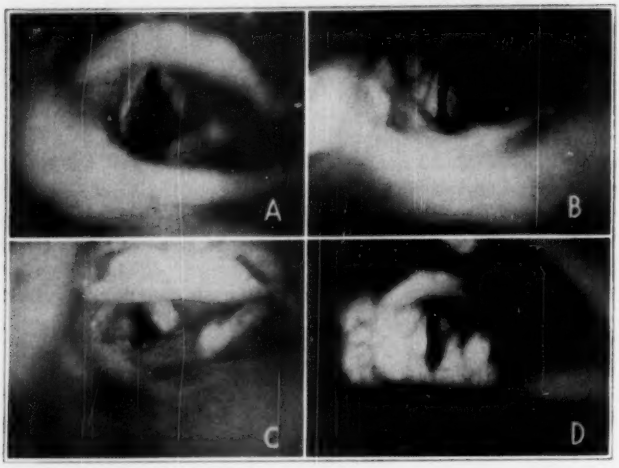

Fig. 2.—Photographs (case 31) taken before (*A*) and after (*B*) daily administration of 1 Gm. of streptomycin for seventy-two days; photographs (case 32) taken before (*C*) and after (*D*) daily administration of 1 Gm. of streptomycin for seventy-eight days.

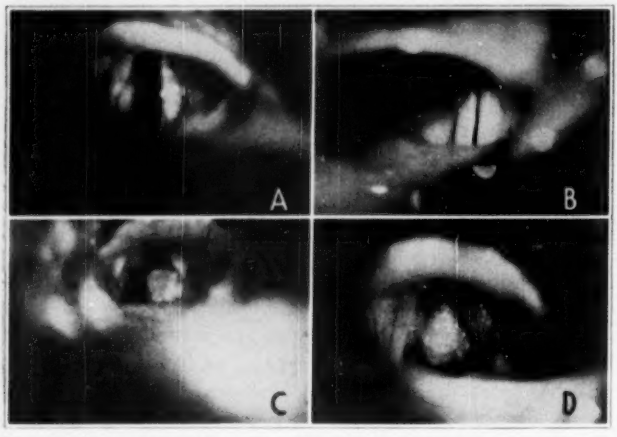

Fig. 3.—Photographs (case 37) taken before (*A*) and after (*B*) daily administration of 1 Gm. of streptomycin for forty-six days; (*C*) photograph (case 33) showing no change in larynx after daily administration of 1 Gm. of streptomycin for four days; (*D*) photograph (case 35) showing no improvement in larynx after daily administration of 1 Gm. of streptomycin for thirty-six days.

One patient (case 28) who had pulmonary tuberculosis, together with what was thought to be a laryngeal extension of the same disease, was treated with streptomycin at home by a private physician. When the patient showed no improvement, he was admitted into the hospital, where biopsy by direct laryngoscopy revealed that this patient did not have laryngeal tuberculosis, as had been suspected, but a malignant lesion of the larynx. This case definitely illustrates the fact that patients with pulmonary tuberculosis may have other types of laryngeal involvement, and it is of the utmost importance that these possibilities be kept in mind in the differential diagnosis. This point is further emphasized in case 35. Histologic examination of the tissue removed from the larynx revealed it to be papillomatous.

Routine examination of the larynx of the patient under treatment is very important in order to evaluate the response to treatment. This can be done in most cases by mirror laryngoscopy without much difficulty. Whenever there is any question about the true diagnosis, direct laryngoscopy and biopsy should be performed.

The larynges of most of these patients were photographed not only before streptomycin therapy was begun but also at intervals during the course of treatment and at the end of treatment. This affords an unbiased evaluation of the response to the therapy. The photographs of the larynx of several of the patients taken before, during and after the streptomycin therapy are reproduced here.

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NOISE-INDUCED HEARING LOSS

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INTRODUCTION

HEARING loss resulting from exposure to noise has received considerable attention in otologic literature.[1] In recent years, this type of hearing loss has assumed increased importance, partly because of its position in theories of audition but principally, perhaps, because industry is coming to realize its occurrence in those members of the labor force who function in a noisy environment. There is a mass of evidence indicating that workers who spend sufficient time in sufficiently high noise levels are exposed to the possibility of incurring an irreversible hearing loss. The length of exposure time and the intensity level of the noise above which loss is imminent are not known within exact limits, nor can the probable severity of hearing loss under given conditions of exposure be predicted on the basis of present knowledge. There appear to be large differences in individual susceptibility to noise-induced hearing loss, such that it is not yet possible to isolate in advance those persons most likely to suffer from exposure to noise.

Reports in the literature have tended to discuss this type of hearing loss on the basis of data collected under two general conditions: (a)

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1. (*a*) Canfield, N.: Trauma from Noise in Industry, Connecticut M. J. **13**:21, 1947. (*b*) Fabritius, H. F.: Some Remarks on Occupational Deafness: A Comparative Investigation, Acta oto-laryng., 1948, supp. 74, p. 140. (*c*) Grove, W. E.: The Noise Hazard, Indust. Med. **18**:25, 1949. (*d*) MacLaren, W. R., and Chaney, A. L.: An Evaluation of Some Factors in the Development of Occupational Deafness, ibid. **16**:109, 1947. (*e*) McCoy, D. A.: The Industrial Noise Hazard, Arch. Otolaryng. **39**:327 (April) 1944. (*f*) McCoy, D. A.: Industrial Noise—Its Analysis and Interpretation for Preventive Treatment, J. Indust. Hyg. & Toxicol. **26**:120, 1944. (*g*) Perlman H. B.: Acoustic Trauma in Man: Clinical and Experimental Studies, Arch. Otolaryng. **34**:429 (Sept.) 1941. (*h*) Peyser, A.: Ueber gewerbeotologische Belastungsprüfungen: Geeignete Frequenz, Acta oto-laryng., 1948, supp. 74, p. 131. (*i*) Rosenblith, W. A.: Industrial Noises and Industrial Deafness, J. Acoust. Soc. America **13**:220, 1942. (*j*) Rüedi, L., and Furrer, W.: Akustisches Trauma und Funktion des Innenohres, Acta oto-laryng. **33**:460, 1945. (*k*) Wilson, W. H.: Prevention of Traumatic Deafness: Preliminary Report, Arch. Otolaryng. **37**:757, 1943.

loss following exposure to noise in industry, military, naval or air services or in fortuitous circumstances, and (b) loss produced experimentally in controlled circumstances.

The first of these conditions usually involves an irreversible hearing loss, that is, one whose extent in decibels does not show any decrease after the person is removed from the circumstances responsible for its onset. The second condition is designed to produce a transient loss, or temporary threshold shift. Such a loss is reversible, that is, it will disappear after a sufficient interval following cessation of the noise stimulus. The same person has been repeatedly exposed to loud sounds in the laboratory without demonstrable permanent decrement in hearing acuity, even after rather severe temporary shifts have been measured.

It does not seem credible—to me, at least—that hearing losses produced under these two different conditions are entirely unrelated. The general law governing the response in any sense organ states that response R is some function of intensity of stimulus I and length of time of exposure to stimulus T.

$$R = f(I,T)$$

In the retina, for example, adaptation to light depends on intensity of the light and length of exposure. Superficially, at least, the temporary threshold shift appears to resemble the same phenomenon.

When temporary shift has been demonstrated, high noise levels have been used; in some instances, these levels approached the pain threshold. I have seen no published report, however, in which high levels have been experimentally applied to human subjects over a period greater in duration than eight hours. Typically, much shorter exposure times have been used, more frequently measured in seconds or minutes than in hours. In terms of the foregoing equation I has been given high values but T has been kept low.

In contrast to this situation, the loss noted in industry most typically follows from a combination of I and T in which both terms have high values. Industrial workers spend years in environments of high noise levels. It is therefore a reasonable inference that a temporary threshold shift represents the initial phase of a process which if allowed to continue produces a permanent decrement in auditory acuity. It is true, of course, that occasionally an excessively high intensity of sound whose duration is very short will produce a severe and irreversible loss. Such losses are seen in members of the armed services, for example, who have been exposed to nearby bomb or shell bursts. This so-called "blast" or "concussion" deafness does not necessarily constitute an exception to the previous statements. Instantaneous pressures generated in such circumstances are so enormous that I in the equation reaches a value in excess of the tolerance limit of the ear; T, therefore, need be very small.

In blast or concussion deafness, the tympanic membrane may sometimes be ruptured; there may also be presumptive damage to the ossicular chain. But histologic evidence from experimental studies of noise trauma in animals leaves little doubt that the important region of damage lies within the inner ear. Actual displacement and destruction of the hair cells are commonly reported observations. There is the further possibility that a temporary threshold shift is partly attributable to central changes; at higher levels, centers of the nervous system may be responsible for inhibitory effects. Much more evidence is necessary before conclusions may be safely drawn about the influence of the central nervous system, but the possibility cannot be ignored.

CHARACTERISTICS OF TRAUMATIC NOISE

The preceding discussion has dealt with the general term "noise" without regard to one of its outstanding features—its energy distribution with respect to frequency. Any sound of sufficient duration can be measured and assigned an intensity value. But its effect on the ear is dependent on the manner in which its energy is concentrated in the audible frequency range.[2] For example, the important energy in typical airplane motor noise is concentrated in the region below 1000 cycles. Frequencies higher than 1000 cycles may, of course, be present but at intensities well below the level necessary to produce hearing loss. In contrast, chipping and riveting hammers making impact against resonant bodies, such as steel boiler plates, may produce high intensity levels in the frequency region above 1000 cycles as well as below. A useful term characterizing the energy distribution of sound with respect to frequency is its "spectrum." The pure tones produced by audiometers and tuning forks are special examples of sounds in which nearly all the energy is concentrated at a single frequency.

There is no single spectrum which describes all noises encountered in the industrial situation. Obviously, the character of the noise prevalent in any plant depends on the nature of the operations in progress there. Until recently, most of the studies dealing with hearing loss as a result of industrial exposure have lacked accurate information about the characteristics of the prevailing noise under investigation. It is now technically possible to analyze the spectrum of complex noise with relative speed and accuracy. In those reports which include statements about the spectrums of the noises studied, as well as in experimental studies in which noise spectrums can be controlled, a general finding has emerged with reasonable consistency. It has been established that noises whose spectrums contain a high proportion of high

2. The effects of supersonic and ultrasonic energies will not be discussed in this paper.

frequency energy effect the ear more seriously than do noises which are predominantly of low frequency, even when the latter noises represent higher intensity levels than the former.

This fact is illustrated in data which I collected during the course of two experimental studies [3] of the temporary threshold shift. In the first of these studies, 4 subjects having normal hearing were exposed to synthetic airplane motor noise. This noise was generated electronically and reproduced by a loud speaker capable of handling high sound levels without serious distortion. The subjects sat in a sound field whose average intensity level at the ear was 112 to 115 decibels above zero reference level (0.0002 dyne per square centimeters). This noise was applied for a period of sixty minutes. Within one minute following termination of the exposure period, audiometric examination was

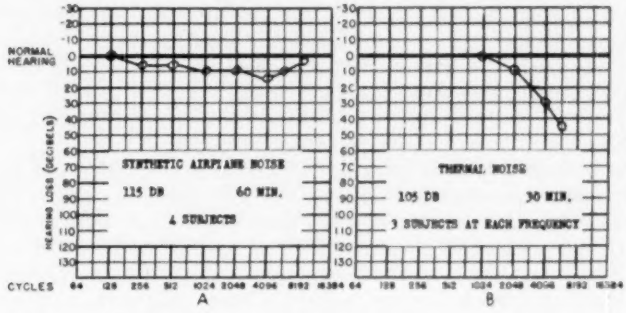

Chart 1.—Average binaural threshold shifts of subjects exposed to (*A*) synthetic airplane noise and (*B*) "white," or thermal, noise.

begun on the subject seated in a standard telephone booth. Frequencies were measured in random order, but 1000, 2000, 4000 and 6000 cycles were always among the first four presented; reasons for this procedure will be discussed later in this paper. Binaural audiograms were taken, the time for each ear averaging approximately twenty minutes. The average binaural shift resulting from these conditions is shown in section *A* of chart 1. Because the effects of this noise were insignificant at low frequencies, a second experiment was undertaken in which study was concentrated on the frequencies 1000, 2000, 4000 and 6000 cycles. The stimulus employed in the second study was so-called "white" noise (thermal noise), a type whose spectrum contains all frequencies [4] at

3. These studies were made in partial fulfilment of requirements for the master's degree in the Department of Psychology, The Graduate College, State University of Iowa.

4. The loud speaker used in this study was not efficient above 5000 cycles.

approximately equal intensity levels. The distinction between this thermal noise and the airplane motor noise previously mentioned is that the former contained important energy components in the high frequency region, whereas the airplane noise did not. The thermal noise was applied for thirty minutes at an intensity level 105 decibels above zero reference. Twelve subjects with normal hearing participated in this study, 3 subjects being measured at each of the designated frequencies. Within one and one-half minutes following termination of the exposure period, audiometric measurement was begun on the subject seated in a sound-treated room. The average binaural threshold shift at each frequency, determined not later than two minutes after cessation of noise is shown in section *B* of chart 1.

Comparison of sections *A* and *B* of chart 1 will show that severer losses are brought about by thermal noise applied only half as long as airplane type noise and acting at an intensity level approximately 10 decibels lower than airplane noise. It should be noted, however, that the greatest threshold shift due to either type of noise falls in the 4000 to 6000 cycle region of the audiogram.

THE ROLE OF VIBRATION

Wherever high noise levels are being produced, vibration may also be present. Its effects have not yet been thoroughly investigated, but some reason exists for the suspicion that vibration may have an influence on the threshold of hearing.

Four additional normal subjects were tested under the conditions given for *A* of chart 1. The platform on which they were seated during exposure to noise was suspended on stiff coil springs and set into vibration by an eccentrically mounted flywheel. Vibration was delivered together with noise for sixty minutes, and the subjects were then measured audiometrically as previously described. The resulting audiograms showed somewhat more threshold shift than that of chart 1*A*, the loss at 6000 cycles being about 5 decibels greater.

It is difficult to quantify the amplitude of vibration experienced by this method, since the excursion of the platform itself does not necessarily represent the effect at the cochlea. Also, vibration may be more directly related to physiologic fatigue than is noise alone. It is possible to state, however, that on the basis of this experiment noise and vibration together produce a greater threshold shift than noise.

THE COCHLEAR REGION OF LOSS

When a hearing loss due to exposure to noise is measured, the frequencies at which the loss is measurable and the degree of severity of loss at these frequencies are dependent on the character (intensity level and spectrum) of the noise. The so-called 4000 cycle dip has

often been discussed by otologists, and some have thought this dip to be diagnostic of noise impairment in the absence of other etiologic factors. Until recently, audiometers were not available with half-octave steps; between 2048 and 8192, the only available measuring frequency was 4096 cycles. It is not surprising, therefore, that hearing loss due to noise often seemed maximum at this frequency. Actually, the true maximum may lie on either side of this frequency and can be revealed only by measurement with a continuous frequency source. It is true, nevertheless, that noise-induced loss does tend to occur most extensively in the higher frequency regions of the audiogram, even when the noise spectrum is composed largely of low frequency energy. It would certainly seem reasonable to conclude that, where the "4000 cycle dip" has been observed, it at least indicated the region of maximum loss to lie between 2000 and 8000 cycles. This statement should not be interpreted to mean that frequencies lower than 2000 cycles are never affected by noise. When ears have been exposed long enough to noises that are loud enough, the loss may spread to involve lower frequency regions of the cochlea; at the same time, the initial high frequency loss becomes severer.

The type of sound most generally employed in the experimental study of the temporary threshold shift has been the pure tone. Investigators have sought to determine a relation between the pure tone stimulus and the frequency region most severely affected. For theoretic reasons, it would be most important if the maximum loss always corresponded to the stimulating frequency, but this exact relation has not been forthcoming. A more frequent observation has been that the loss seems maximum at a frequency level one-half to one octave above the stimulus frequency; even this finding is subject to variation. When pure tones produce a temporary threshold shift, the loss still follows the general rule: Low frequency tones tend to induce losses at higher frequencies.

RECOVERY

When an ear has been removed from a noisy environment, the decrement in hearing acuity resulting from noise tends to diminish with passing time. In other words, the audiogram shows a decrease in the initial loss. For temporary threshold shifts in human subjects, a return to normal threshold is the primary requisite without which the experiment could not safely be undertaken. Dependent on the degree to which the shift is extended (number of decibels temporary loss), recovery takes place with time in a manner which will be illustrated in a later section of this paper.

The factor of time cannot be so readily controlled when loss due to industrial noise is studied. In the past, such studies have usually stressed the already existing loss found among workers whose time of

service, hence exposure, differed from individual to individual. Preemployment audiograms were not available; consequently, the status of the worker's hearing prior to exposure was not known to be normal. In at least one such study,[11] the effect of removal from the noisy environment showed that some recovery took place in workers whose audiograms might otherwise be assumed to represent permanent loss. This does not mean that hearing necessarily returns to normal when workers are removed from noisy operations. There can be no doubt that, for a given person, some critical exposure time and intensity level exist which when exceeded will produce a permanent residual loss. The point is, rather, that the true residual loss can be determined only after it has been stabilized by removal from noise for a sufficient period. Insufficient data have been collected to permit a generalization about the length of time necessary to achieve stability, but a minimum of forty-eight hours [5] certainly seems indicated; even longer times may eventually prove to be necessary. Prevacation and postvacation audiograms suggest themselves as one practical way of collecting more data when the residual loss is to be determined. The shift of workers from noisy to non-noisy operations within a plant might also offer opportunity for more information on this matter. It must be recognized here that conditions for research in industrial hearing problems have not been ideal, but it is to be hoped that the near future will bring a change as the result of the common aspiration of all parties concerned to reduce the incidence of traumatic hearing loss.

It has also been shown that hearing loss increases during the working day in a noisy environment. Thus, it is probable that some recovery has taken place during the interval following the previous working day, even in those workers displaying a relatively severe loss. The question of true residual loss assumes even greater importance if one accepts Rosenblith's [11] observation (shown also in audiograms by McCoy [16]) that a normal ear exposed for the first time to industrial noise conditions may undergo a severer temporary threshold shift than the hearing loss found among the personnel employed at the same location. It is somewhat difficult to interpret this finding. On the one hand, it might represent some relatively simple process of adaptation in the sensory end organ; on the other hand, it might mean that the process is complicated by central nervous system changes of a much higher order.

Whatever its cause, loss due to noise must be considered with reservations imposed by the temporal relations previously cited. In experimental studies of the temporary threshold shift, control of time

5. This is the minimum recommended by the Sub-committee on Industrial Noise of the American Academy of Ophthalmology and Otolaryngology.

should be a matter of primary importance. The typical technic employed in such studies has been exposure of the ear to sound at known intensity levels for a specified length of time, then measurement of the complete audiogram at stated intervals following termination of the sound. This procedure yields an audiogram composed of thresholds actually measured at different times as reckoned from the termination of the stimulus, since the taking of a complete audiogram cannot be done instantaneously. Depending on the number of frequencies included and the standard of accuracy required, a complete (binaural) audiogram may take from five to twenty minutes. Within twenty minutes, an important amount of recovery may occur at certain frequencies.

A more accurate method of controlling the time factor in experimental studies requires that measurements of recovery be confined to a single frequency following exposure. This method is admittedly less efficient, since it requires more subjects and more time to permit the accumulation of the same amount of data than does the method involving the complete audiogram on each individual. It may be argued, however, that experimental studies should aim for accuracy. In view of the general agreement that low frequency regions of the audiogram show more resistance to temporary threshold shift, it seems reasonable to concentrate future experimental studies on the critical high frequencies, thus lessening the amount of work required.

THE TEMPORARY THRESHOLD SHIFT

Initial Extent.—The experimental arrangements for the collection of the data presented subsequently have been described in the discussion of section *B*, chart 1. Data for the 1000 cycle frequency have not been included, since the shift at this frequency was too slight to be of significance.

Chart 2 shows a bar graph illustrating the initial shift at 2000, 4000 and 6000 cycles. At each frequency, the shifts for 3 subjects are given. Each shift is the mean of three exposures per subject. Subject 7, for example, was exposed to a thermal noise on three different days. His average loss measured within two minutes after removal from noise is seen to have been 40 decibels at 4000 cycles.

Inspection of chart 2 will indicate the differences among subjects in susceptibility to threshold shift for a given frequency. These differences occur, not only from subject to subject, but also between two ears in the same subject, as will be shown later.

Recovery with Time.—The general form of the recovery curve is shown in chart 3. This figure gives the course of recovery for 2000, 4000 and 6000 cycles as measured on 9 subjects, 3 subjects at each frequency. Each experimental point is the mean of nine postexposure

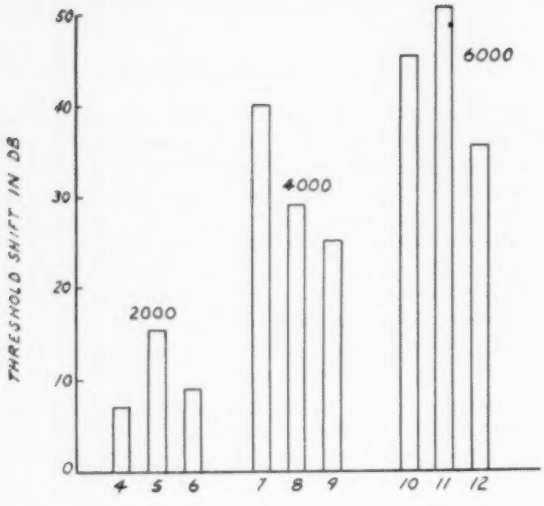

Chart 2.—Differences among subjects in susceptibility to threshold shift for a given frequency.

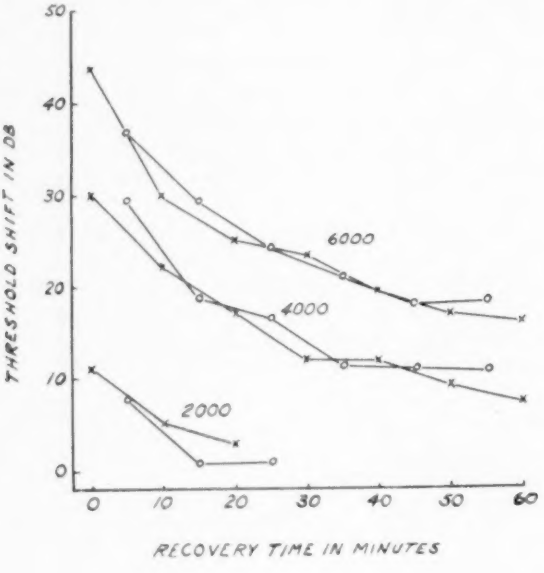

Chart 3.—Recovery curves for 9 subjects (3 for each frequency). *X—X* indicates the curve for the left ear; *O—O*, the curve for the right ear.

thresholds. These measurements were made at five minute intervals, alternately between left and right ears. The initial shift was always measured on the left ear.

It may be seen that the course of recovery is not a linear function of elapsed time. The recovery rate is rapid at first but tends to slow up. In terms of these curves, several hours must necessarily have intervened before acuity at these frequencies returned completely to normal, although at 2000 cycles there was so slight a shift that recovery was virtually complete after twenty-five minutes as shown.

When the data of chart 3 are analyzed into those for the individual subjects, the recovery curves shown in chart 4 result.

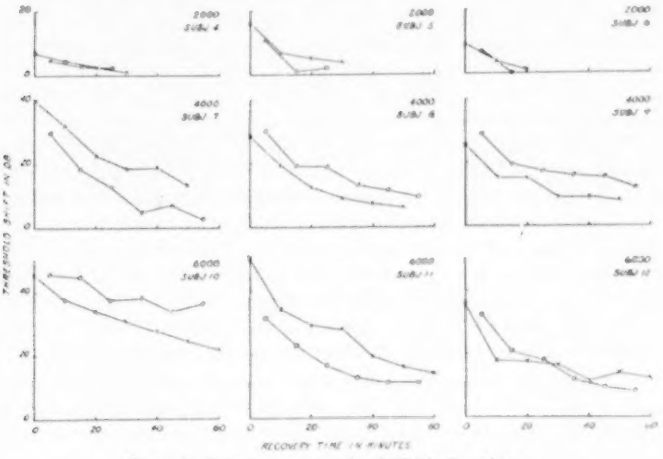

Chart 4.—Recovery curves for individual subjects.

It will be noted that, even in the same subject, ears show differences in susceptibility to noise-induced loss. Another important detail shown by these curves is the difference among subjects in rate of recovery. Subject 11, at 6000 cycles, had an initial shift of 50 decibels measured on the left ear; sixty minutes later, the loss was reduced to 14 decibels, a recovery of 36 decibels. Subject 10, at the same frequency, showed an initial shift of 42 decibels; sixty minutes later, the loss was still 22 decibels, a recovery of 20 decibels.

PREDICTIVE OR "PREMONITORY" TESTS

I feel that there is a definite need for a test or a battery of tests which could be applied prior to the employment of a workman in a noisy environment. Such a test would be based on the assumptions (1) that

there are noise-susceptible ears, meaning certain persons more vulnerable to noise-induced hearing loss than others, and (2) that it would be possible to select out the susceptible person by tests given in advance of industrial exposure. The alternative to preliminary testing is the retesting of an employee after a given time of exposure to the actual noisy environment, with a given recovery period intervening. This procedure is unquestionably a highly desirable one; in certain cases, however, it might depend for its effectiveness on the production of a residual loss which might be avoided if a practical preliminary test can be devised and validated. There now appear to be good grounds for belief that, in some persons, an important residual loss occurs early in the exposure period, although it has not yet been suggested that serious loss may become permanent in so short a time as one week under ordinary industrial conditions. MacLaren and Chaney,[1d] studying employees working in noise levels approximating 105 decibels, stated that most of the observed loss was established during the first three or four weeks of exposure, with subsequent very gradual change; there was a tendency for loss to reach a limiting value early in the course of exposure. Fabritius [1b] set a somewhat longer limit as a result of his study of quarry workers but said that "grave impairment occurs during the first years." Although, in general, industrial noise probably does not often reach the traumatic levels of certain service conditions, I have seen more than one serviceman with a severe residual loss induced by one or two days on the firing range, especially those employing the 50 mm. machine gun. Until more data have been collected under controlled conditions, the possibility of sudden onset and severe residual loss cannot be ignored.

It might also be supposed that industry would welcome a valid predictive test. As a quick test, it would reduce the retesting load. As a reliable test, it could serve to reduce the number of claimants for compensation by eliminating suceptible persons in advance of damage.

Characteristics of a Practical Predictive Test.—Practical predictive tests are yet to be developed. It seems probable, however, that the direction of research leading to a predictive test is now indicated.

1. The test itself must not be time consuming to a point inconsistent with the medical examination of new employees.

2. It must be a test capable of administration and interpretation by operators not highly trained.

3. The apparatus required must be as simple as possible.

4. The test should probably be confined to the high frequency region of the audiogram.

5. The stimulus should probably be a complex noise having a thermal, sawtooth or spike spectrum, generated electronically and presented on receivers.

6. The criteria of susceptibility will probably be found to be both degree of initial shift and rate of recovery. It is a reasonable inference that persons showing large initial shifts and slow rates of recovery may be persons susceptible to early impairment under industrial conditions.

7. The test, once developed, must be validated by application in the industrial situation.

Wilson [1k] has advocated the use of a predictive test, and Peyser [1h] has stated that he is employing a predictive test in an attempt to isolate in advance the noise-susceptible ear. These investigators use pure tones as the noise stimulus which produces the temporary threshold shift; they consider the extent of the shift in decibels to indicate susceptibility when the shift exceeds a certain criterion value. It is evident that progress is being made toward the development of a suitable test. In my opinion the two greatest needs at the moment are as the following:

1. Standardization—tests should employ reasonably comparable presentation procedures, terminology and criteria.

2. Validity—the usefulness of predictive testing rests on the clinical accuracy with which susceptible persons can be isolated.

TRANSTYMPANIC TECHNIC IN SURGICAL OTOLOGY

Transtympanic Postscripts

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THE TRANSTYMPANIC technic may be regarded as a new method in surgical otology. Its object is to make directly accessible the tympanic structures anterior to the mastoid process, as well as the mastoid process and its contents. The anterior structures are the attic, aditus, tegmen, vestibuloampullary region, facial canal, round window, promontory, tympanum and hypotympanum, as well as the forward extension of the hypotympanum into the eustachian tube and the muscular canal containing the tensor tympani. The posterior structures include the mastoid process, the genu and the descending part of the facial canal, lateral sinus, semicircular canals and the tegmen.

Certain procedures are greatly facilitated by the use of the transtympanic approach, and, indeed, some of them can be carried out only by this route. Details of transtympanic atticoantrostomy and fenestration have already appeared in appropriate journals; publication of the other procedures will follow.

PROCEDURES FACILITATED BY THE TRANSTYMPANIC APPROACH

1. Atticoantrostomy
2. Fenestration
3. Radical mastoidectomy
4. Exposure of the facial canal from the processus cochleariformis to the stylomastoid foramen
5. Operation on the hypotympanum, with special reference to restoration of hearing in the chronically infected ear
6. Decompression operation for Ménière's syndrome
7. Operation for drainage and exploration of the petrous tip

FUNDAMENTAL TECHNICAL PRINCIPLES

Those familiar with previous publications on the transtympanic approach will recollect that this technic pivots on the following fundamental technical principles:

To work from the front backward, "square on," to the operational field—the frontal attack on the outer wall of the attic and the tympanic contents.

From the Department of Surgery, University of the Witwatersrand.

To use the meatal corridor in place of the exenterated mastoid process as the approach to the tympanic structures.

To fashion the tympanomeatal flap or an appropriate cutaneous plastic flap, depending on the procedure contemplated, at the very outset of the operation, and to protect such flaps with certainty.

To expose tympanic structures directly by removal of the outer attic wall and to extend such removal to the posterior bony wall of the meatus and the mastoid cortex, where the posterior structures are to be explored.

To expose the outer free edge of the tympanic plate and, where wider access to anterior tympanic structures is desirable, to ablate this edge subcutaneously and subperiosteally as far down as may be necessary.

To detach the meatal cartilage from the tympanic plate as well as from the roof of the bony meatus, with either the antetragal or endaural incision. (The soft tissues are mobilized from their bony base, and suitable retraction exposes the meatal corridor and the edge of the tympanic plate.)

In two recent publications, Lempert [1] discusses his use of the transtympanic approach to achieve immediate exposure of the lateral attic wall, using the meatus as the corridor. In his decompression operation for Ménière's syndrome, a portion of the attic wall is removed to expose the stapes. In the mastoidotympanectomy the entire wall is removed to reach the mastoid antrum as an alternative to the blind search for this vital tympanic landmark through a sclerotic mastoid cortex. It is gratifying that the transtympanic approach should gain the support and approval of this distinguished authority.

TRANSTYMPANIC ENDAURAL INCISIONS

My first efforts in transtympanic surgery were directed toward the endaural incision. Fear of meatal atresia, massive displacement of tissue and destruction of the circumferential integrity of the meatus lent a certain bias toward the antetragal incision. These drawbacks have been overcome, and the endaural incisions to be described here will be found efficient for all transtympanic operations.

In contrast to the Lempert endaural incision, the transtympanic endaural incision begins at and hugs the anterior aspect of the meatus. The improved transtympanic endaural incision not only possesses the merit of great simplicity but, what is equally important, possesses very obvious landmarks and a capacity for accurate description. The time required for immediate exposure of the outer edge of the tympanic plate or the anterior bony wall of the meatus is a matter of seconds. This plate can be reduced subcutaneously and subperiosteally by a nibbling forceps and a curet to an extent necessary for access to the tympanic or hypotympanic structures—a useful step when the meatus is narrow.

1. Lempert, J.: Lempert Decompression Operation for Hydrops of the Endolymphatic Labyrinth in Ménière's Disease, Arch. Otolaryng. **47**:551 (May) 1948; Lempert Endaural Subcortical Mastoidotympanectomy for the Cure of Chronic Persistent Suppurative Otitis Media, ibid. **49**:20 (Jan.) 1949.

In fenestration procedures it is generally not necessary to ablate the tympanic plate except where it is attached to the bony roof of the meatus and where, occasionally, it flares out and overhangs the meatal canal. Its removal subcutaneously presents no difficulties; a curet generally suffices.

Basic Endaural Incision.—The basic incision, as its name implies, is used in every transtympanic procedure. Descriptions of incisions are unnecessarily difficult and confusing when the main step is not kept segregated from the subsidiary ones. Accordingly, the auxiliary

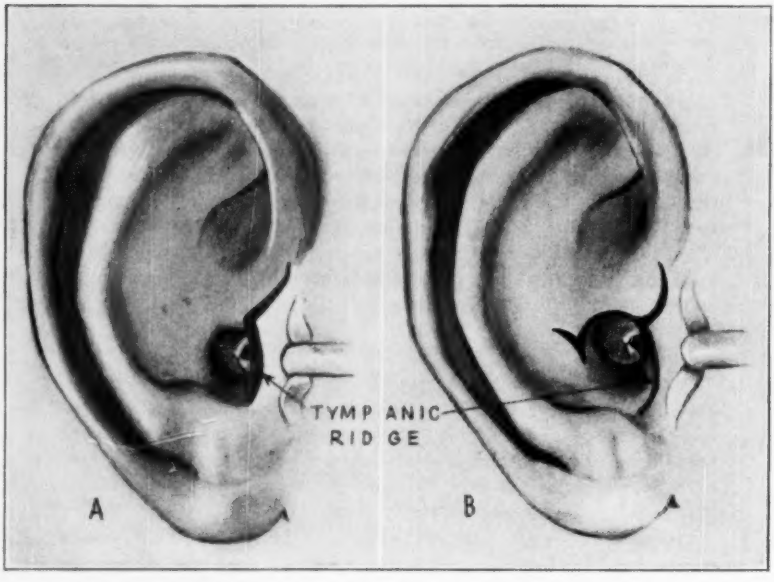

Fig. 1.—*A*, basic transtympanic incision, hugging the anterior aspect of the meatus. The internal or meatal part of the basic incision is down to bone—the tympanic plate (anterior bony wall of the meatus) and the bony roof; the outer portion of the basic incision as it leaves the meatus is down to temporal fascia only. *B*, auxiliary incisions. The main auxiliary incision is made down to bone and runs across the upper half of the bony meatus at the level of the free edge of the tympanic plate. A small gusset at its posterior extremity will allow the skin to adapt itself to the contour of the widened bony meatus.

incisions suggested for the various transtympanic operations are dealt with separately.

The basic incision is completely extracartilagenous and traverses three portions of the temporal bone: (*a*) the "tympanic ridge"; (*b*) the roof of the bony meatus, and (*c*) the zygomaticosquamous area (the groove between the helix and the tragus).

"Tympanic Ridge" Section: The meatus is spread with a short Killian or similar speculum, the tragus being everted in a forward direction. A bony ridge is immediately exposed in the anterior segment at a depth of about 1.0 cm. This superlatively clear landmark may conveniently be called the "tympanic ridge." It does not appear to have been given a name. It is formed by the outer free edge of the tympanic plate (pars tympanica ossis temporalis) and the anterior bony wall of the meatus, to which is attached the meatal cartilage. In order that this landmark should not be distorted or obscured, it is recommended that infiltration of the soft tissues be delayed until after completion of the tympanic ridge incision, when general anesthesia is employed.

The incision is made along the tympanic ridge, from below upward, down to bone. To be quite sure of incising extracartilagenous material, I place the incision slightly medial to the ridge, that is about 1.0 mm. nearer to the tympanic membrane. Infiltration of the meatal and contiguous soft tissues now takes place.

Meatal Roof Section: From the upper limit of the tympanic ridge, the incision is continued, down to bone, along the roof of the bony meatus, with the operator detaching the meatal cartilage from this region. The incision thus far is an inverted L.

Zygomaticosquamous Section: The cut is then continued down to the temporal fascia only, with the operator rounding the zygomatic root to end externally on the squamous temporal area between the helix and the tragus. Viewed laterally, this composite basic incision is almost a straight line; viewed anteroposteriorly, it has a sinuous outline. Very little departure from directions is permissible in the basic incision; the auxiliary incisions are more flexible in this respect.

Auxiliary Incisions.—For the various transtympanic procedures, appropriate auxiliary incisions must be made. These incisions run across the roof or posterior wall of the bony meatus at such levels and in such directions as to expose the structures desired to be reached.

For fenestration, the auxiliary incision runs across the roof for about half the circumference of the meatus at the level of the upper limit of the tympanic plate. This auxiliary incision also constitutes the upper incision of the eventual tympanomeatal flap. The main auxiliary incisions for the other procedures are indicated in figure 1 *B*. As has been pointed out in previous publications, these incisions are flexible and may be altered to suit the surgeon's particular requirement. The basic incision, however, should retain its simplicity; it requires no elaboration.

When the auxiliary incision, which also is made down to bone, has been completed, two flaps have been created. The posterior one is L shaped, and the anterior one is, more or less, bounded by a straight

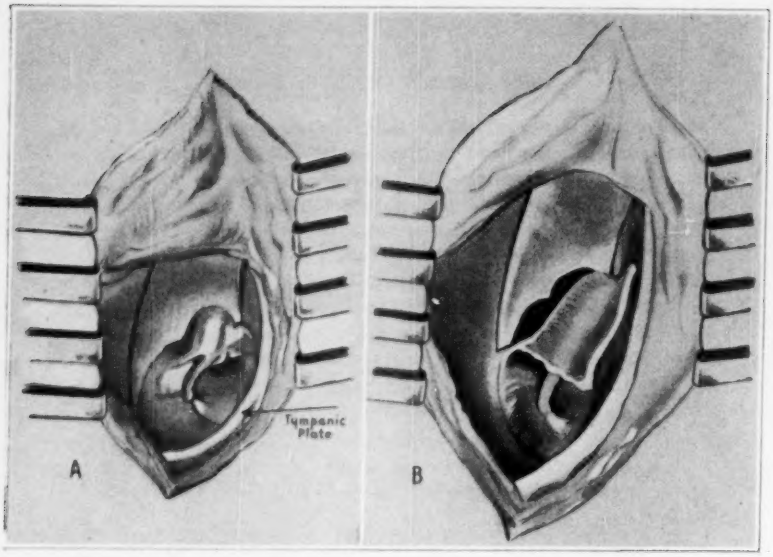

Fig. 2.—*A*, further auxiliary incisions, either for the tympanomeatal flap or to provide cover for the atticoantrostomy cavity. The tympanic plate edge is taken down to demonstrate wide tympanic exposure. *B*, auxiliary flap reflected from the bony meatus. The annulus tympanicus and spina tympanicus are revealed.

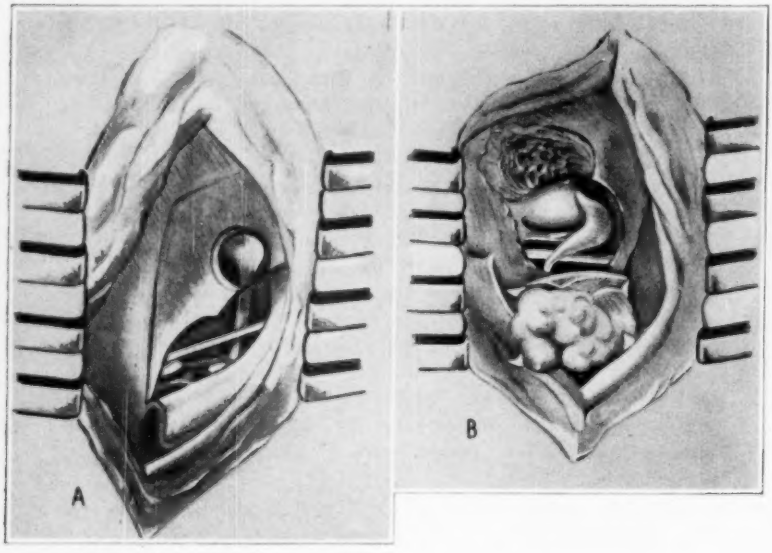

Fig. 3.—*A*, small opening made at the anterior margin of the annulus to reveal the body of the incus. The meatal flap is reflected to show the long process of the incus, the chorda tympani, the stapedial tendon, etc. *B*, removal of entire attic wall to expose the atticoantral region. The incus is still in situ. The flap is protected by packing and wax.

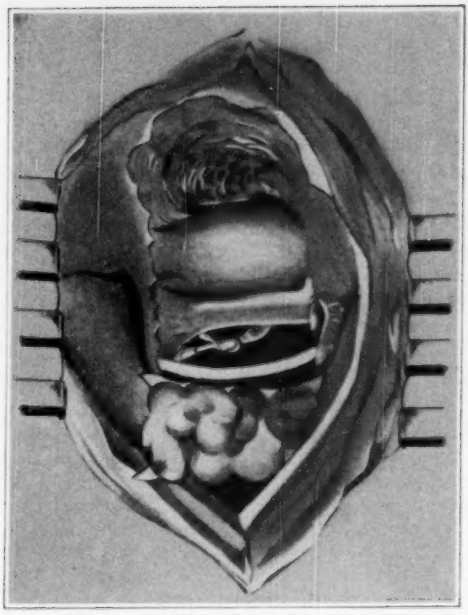

Fig. 4.—After removal of incus. From above downward are exposed the atticoantral region, "dome," facial nerve, eminentia pyramidalis, stapedial tendon and stapes, chorda tympani and head of malleus. With the facial nerve in full view, the mastoid cavity can be exposed by further removal of posterior bony meatus and mastoid cortex. In sclerotic cases extensive exenteration is rarely necessary.

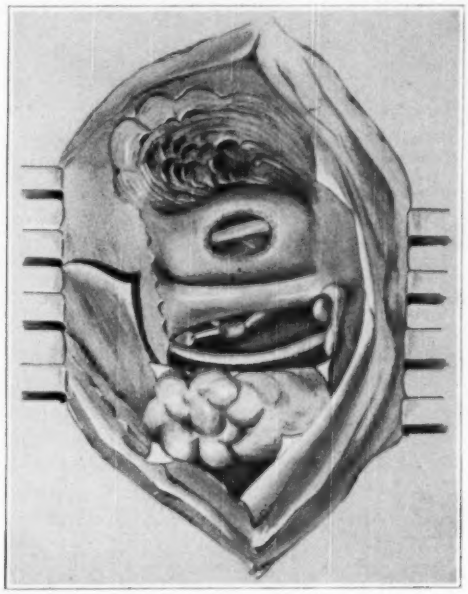

Fig. 5.—Illustrations of atticoantrostomy and fenestration. The rim of the fenestra is lead burnished.

line—the basic incision. With a periosteal elevator the posterior flap is now raised from the underlying bone. The anterior flap is mobilized from the root of the zygoma and from the tympanic plate. This gentle forward maneuver detaches the meatal cartilage and sweeps it from the edge of the tympanic plate in a forward direction and also sweeps it from the roof of the bony meatus. After this subperiosteal elevation, a self-retaining retractor is inserted and opened out.

The meatal corridor is now revealed with the tympanic membrane, and from behind forward are also revealed Henle's spine, with some of the mastoid cortex overlying the mastoid antrum, the lateral edge of the superior bony meatus and the root of the zygoma. It is useful at this stage to bevel down this overhang formed by these three bony landmarks, especially when it is intended to use the operating micro-

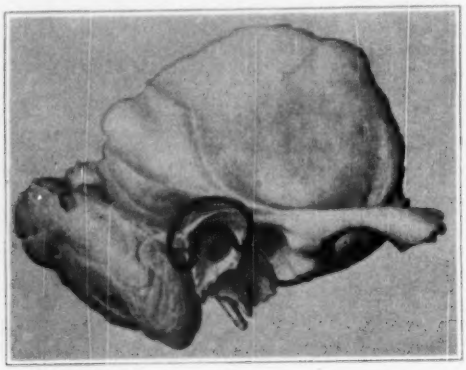

Fig. 6.—Beveling the bony overhang of the meatus. The kidney-shaped area marked on this temporal bone is beveled off with a steel cutting burr with a large diameter. The area includes, from behind forward, a little of the mastoid cortex, the suprameatal spine, the outer edge of the bony meatus and the root of the zygomatic process. The free edge of the tympanic plate anteriorly is also taken down subcutaneously, should it overhang the meatal opening. It will be noted that by removing the posterior bony meatus and mastoid cortex, the mastoid contents, including the facial canal, etc., may be exposed and explored and a transtympanic atticoantrostomy or radical mastoidectomy carried out under perfect visual control. (After Sobotta, R. H. J.: Atlas und Lehrbuch der Histologie und Mikroskopischen Anatomie des Menschen, ed. 2, München, J. F. Lehmann, 1911.)

scope. For this purpose a spherical steel cutting burr with a large diameter is employed. This burr creates a funnel-shaped entry to the rest of the meatus. The subsequent maneuvers, such as the fashioning of the tympanomeatal flap and the taking down of the lateral attic wall, proceed as already described.

The bony edge of the tympanic plate is also exposed on retraction. When the view of the tympanic membrane is restricted, owing to a

narrow meatus, the tympanic plate is nibbled down (subcutaneously and subperiosteally) until exposure is satisfactory.

It has been mentioned that certain objections were encountered when an endaural incision was originally used for the transtympanic approach. The first of these objections was the destruction of the circumferential integrity of the meatus. This objection is overcome by careful suturing of the flaps after completion of the operation. The sutures should be placed according to the directions that follow: one suture at the outer edge of the external auditory meatus, so as to conserve this ring, and another at the point just where the incision leaves the meatus and reaches the space between the helix and the tragus. These two sutures preserve the meatal integrity and appearance. The large, unsightly opening that is frequently encountered in cases of endaural mastoidectomy is avoided.

PACKING METHODS

Some workers use a most exacting ritual; others, notably Professor Lund, of Copenhagen, use the simplest possible method and achieve uniformly excellent results. Professor Lund packs ¼ inch (0.5 cm.) ribbon gauze soaked in penicillin-sulfathiazole solution over the skin covering the fenestration and into the attic space. I use a slight modification of this method. It is desirable to dimple the skin into the fistula to insure contact between it and the membranous canal.

Recently I have had occasion to use a special packing material, marketed under the trade name of "calgitex," which is a seaweed preparation impregnated with antiseptic material. It is supplied in ribbon form. The advantage in using this material is that it forms a homogenous plastic mass, which is closely adapted to the tympanomeatal flap and the fenestration. At its periphery and exposed surfaces, it undergoes very slight and slow gelatinization.

My practice now is to powder liberally the tympanomeatal flap and the attic cavity with penicillin and sulfadiazine and then gently but firmly to pack the "calgitex" ribbon so that it becomes closely adapted to the flap with the underlying fenestration and the attic walls. No packing is placed over the tympanic membrane.

An oversized piece of viscose sponge, about one and one-half times the diameter of the meatus, is inserted into the meatal opening in such a manner as not to press on the drum but merely to keep the meatus gently spread. The "calgitex" ribbon, incidentally, is also packed onto the posterior skin flap so as to adapt it closely to the attic or antral wall.

The outer meatal sponge is removed on the fourth or fifth day together with the sutures. The "calgitex" packing is left for another two or three days. By virtue of the gelatinization on the peripheral

surfaces of the packing, its removal is entirely painless and can be done without an anesthetic and without fear of detachment of or injury to the flaps. These advantages are a great comfort to both patient and surgeon. Another advantage of this two stage packing is that improvement of the patient's hearing becomes very evident when the meatal sponge pack is removed.

It is my belief that the requirements desired of a packing material—close homogenous adaption without adhesion to the flaps and, at the same time, without acting as an impenetrable barrier to normal post-

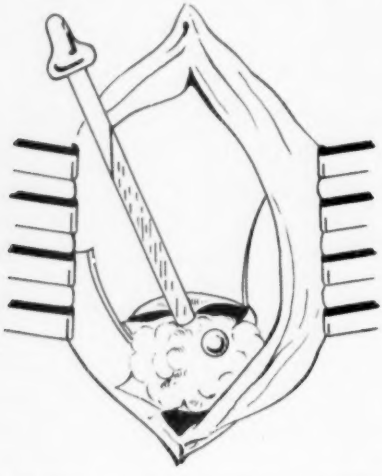

Fig. 7.—Molten flexible dental wax deposited on the protective cover over the tympanomeatal flap.

operative serous discharge—are approximated in the "calgitex" material.

PROTECTION OF THE TYMPANOMEATAL FLAP

It has already been described how the tympanomeatal flap in the transtympanic technic is safely tucked under the ledge of the tympanic plate at the outset of the operation. The flap is covered with a little gutta percha tissue and, for additional safety, packed off with pledgets of cotton wool. This procedure is entirely adequate, and injury either to the flap or to the drum when these directions are observed is rare. Occasionally, a fiber from the cotton wool pledgets invades the operative field, in which case the burr will pick up the entire pledget of cotton wool, which then must be replaced. To overcome this minor nuisance,

I am now using a simple wax technic for good measure, which I am sure my colleagues will find useful.

A few drops of melted wax used for dental impressions are dropped on to the pledgets of cotton wool, which cools rapidly to a soft, flexible consistency. The mass is molded or plastered down from the tympanic ring outward so as to form a smooth homogenous surface shelving inward. Accidental instrumentation toward this wax wall will dis-

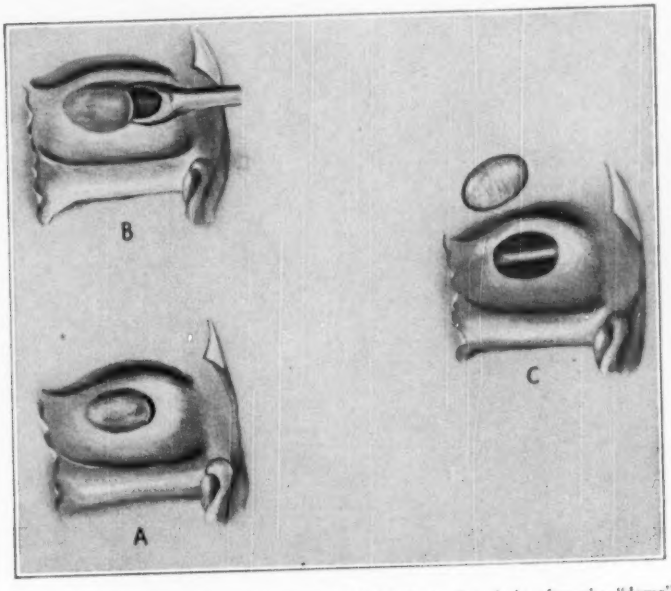

Fig. 8.—Diagrammatic representation of the "scoop" technic after the "dome" wall has been thinned down anteriorly, superiorly and laterally. Width of fenestra is more important than length.

tribute the force equally in all directions, and it may safely be said that any danger of injury to the drum or tympanomeatal flap is banished.

BONE DUST–FREE UNCAPPING OF THE LABYRINTH

Lempert has described a dust-free uncapping technic. The "dome" is ground down until a cupola of endosteal membrane is exposed. This membrane is circumferentially incised and lifted out as a cap. All bone dust and spicules are carefully removed and the area irrigated before the uncapping. I have attempted this maneuver on more than 50

domes, and my difficulty has been that before the entire endosteal cupola is prepared there already is, on too many occasions, a pinhole opening into the perilymphatic space. Such a premature opening negates the object of the bone dust–free technic. The circumferential incision of the endosteum also has occasioned me much nervousness from fear that I might injure the membranous labyrinth. I have found it very difficult to get such a precise oval surface, as has been described, of endosteal membrane exposed. For those surgeons who share my

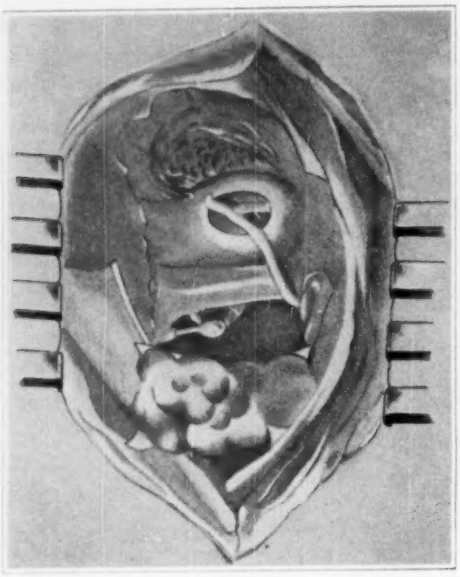

Fig. 9.—Chorda tympani insert (Modified after Rosen). The chorda tympani nerve is mobilized and about to be inserted into the fistula. (This drawing was made from a dissection which I prepared.)

Rosen's chorda tympani technic: It is too soon for me to venture an appraisal of this promising procedure. I have used it in a series of cases and have been most favorably impressed. It is my belief that the future will show that Rosen's technic is a real contribution to the battle to keep the fistula open. There are three potent reasons why Rosen's work should be entitled to support and a thorough trial: 1. He has conceived the idea of a living pedicle graft in close proximity to the fistula. 2. This pedicle is a nerve trunk, a tissue which is very resistant to invasion. It would act as an obturator and may be assumed to counteract osteogenetic encroachment in the fistula. 3. It acts as an intervening member between the skin of the tympanomeatal flap and the membranous canal.

Much convincing literature has appeared of late demonstrating the desirability of securing contact between the membranous canal and the tympanomeatal skin, resulting in maximal and lasting improvement in hearing. By the interposition of this small nerve pedicle, which has its own blood supply and whose viability is therefore assured, such contact is made doubly certain.

preoccupation with and fear of instrumentation within the lumen of the fistula, I recommend the following alternative technic:

The dome is ground down laterally, anteriorly (very simple with the transtympanic approach) and superiorly until not only a double gray line appears but, where these two lines are joined anteriorly, a gray crescent is formed. This grinding down does not quite reach the endosteal membrane—a thin layer of bone still overlies it. The anterior portion of this crescent is ground down a little deeper so as to provide purchase for a cutting instrument presently to be used. On some domes a gray piriform area appears, with the stalk pointing posteriorly, instead

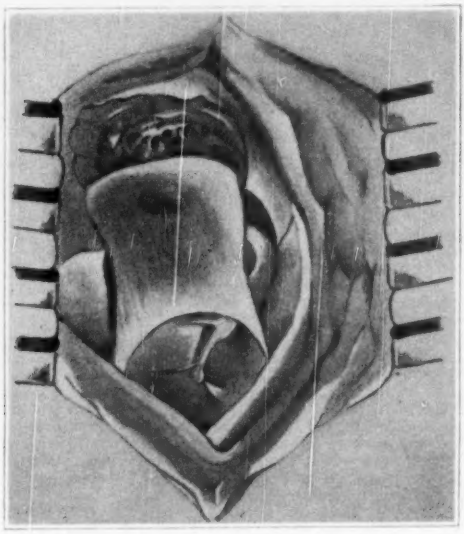

Fig. 10.—Replacement of flap. Head of malleus is shown, but when it embarrasses the smooth adaptation of the flap, its removal is advised.

of the double gray line. With a 00000 Lempert curet, which is especially sharpened for this purpose, the next step is executed.

UNCAPPING THE LABYRINTH

The sharp scoop is engaged anteriorly and cuts through the thinned endosteal bone, and the whole spoon is lifted in a posterosuperior direction. It cuts through the thinned dome wall as well as the attached endosteal membrane. At the posterior limit of the gray lines, the spoon is levered outward so as to fracture the denser bone overlying the lateral canal. This maneuver avoids damage to the canal, which at this location issues from its minute bony enclosure. The shape of the fistula

is piriform, and the surgeon will be astonished by its size. In a previous publication, I described a cistern of perilymph at the anterior location of the dome. This cistern forms a liquid cushion protecting the mem-

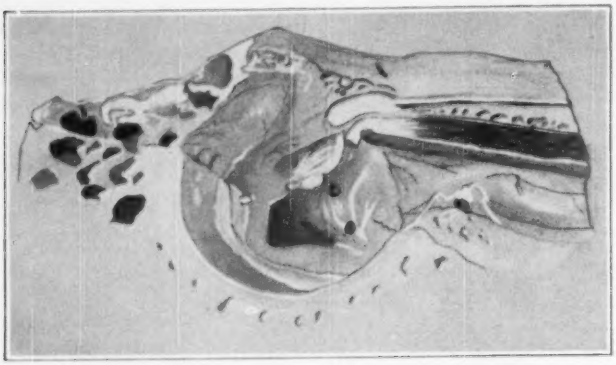

Fig. 11.—Dissection and diagram illustrating the rationale of Popper's operation for Ménière's syndrome. The upper perforation of the promontory invades the scala vestibuli and drains its perilymph. A small Shambaugh hook is introduced through this opening to tear the membranous sac of the caecum cochleare and, possibly, the saccule and utricle. This tear releases the endolymph. The upper perforation is made in line with the upper curvature of the round window; the lower perforation on the promontory is made in line with the lower curvature of the round window and drains the perilymph of the scala tympani. The promontory is exposed by reflecting the drum forward with an auxiliary incision around the posterior aspect of the meatus, close to the annulus. The basic incision and retraction described will be found adequate. There is no need to remove any part of the outer attic wall or extract the stapes. This drainage of the perilymph of the scalae as well as of the endolymph (by tearing the caecum cochleare) has been found successful in controlling vertigo and intolerable tinnitus in 2 cases in which this simple operation had been performed. After the promontory has been dealt with as described, the middle ear is dusted with penicillin and sulfadiazine powder. The drum is replaced; the meatus is gently packed with penicillin tulle. One stitch at the outer margin of the meatus generally suffices.

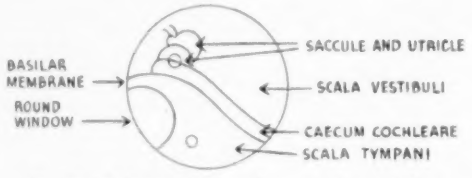

Fig. 12.—Schema of internal aspect of promontory.

branous labyrinth. It is therefore perfectly safe to cut through anteriorly without the slightest risk of damage to the membranous labyrinth. However, this does not apply to the posterior procedure, and hence this edge is fractured outward. Needling shreds of endosteal mem-

brane or instrumentation within the lumen of the fistula is rarely necessary. After irrigation the canal will be found intact under the operating microscope.

According to Holmgren, it is immaterial whether the fenestration is performed by the endaural technic of Lempert, the postaural technic of other workers or by the method of Popper. Holmgren, however, emphasizes that the importance lies in the size of the fenestra.[2]

I have always maintained that the size of the fenestra is its first defense against osteogenetic closure. By the maneuver described here, a maximal fistula is achieved. Some cadaver practice with this technic will convince those who would care to try it. A large fistula may be the cause of more intensive and prolonged postoperative vertigo, but its advantages outweigh such transient distress.

CONTINUOUS SUCTION IRRIGATION

Some operators make a great feature of suction irrigation during the fenestration maneuver. I have found that bone dust is too adherent to be removed by this gentle flow of fluid. Lempert uses an ordinary inexpensive dental chip syringe filled with isotonic sodium chloride solution and directs the stream, under perfect visual control, all about the field every few seconds during the fenestration maneuver. The force of the stream is controlled by the pressure on the bulb, and the field is cleared in minimal time of every vestige of bone dust and spicules. Having used both continuous suction irrigation and the chip syringe, I find the syringe more effective than the suction irrigation, which does not dislodge adherent particles. Moreover, Lempert's method has the benefit of great economy of space in the operating field. I keep two or three such dental chip syringes filled with isotonic sodium chloride solution in front of me, and they are constantly refilled. The suction apparatus is operated by the free hand or, alternatively, the tiny suction tube may be clamped into such portion of the field that it will effectively clear of debris. Another minor point is that the fluid

2. Holmgren, G.: Present-Day Surgery of Otosclerosis, Acta oto-laryng., 1948, supp. 74, p. 19. In this paper, Holmgren made the following statement: "It is not Lempert's endaural method which is decisive, even if this has advantages. It is equally possible to use the retroauricular incision or, as I have done in several cases, to enter via the posteriorly widened auditory canal, without curetting the mastoid, or—as Popper does—to enter via the anterior aspect of the external auditory canal. Obviously also, it is not the performance of the operation with burr and spoon instead of chisel and mallet which is of prime importance, any more than irrigation and suction, which is used by Lempert instead of the usual drying method. The most important point is the following: the new fenestration is made on the dome of the vestibule. . . . At this site the new fenestration can be made larger than at any other point in the vestibular apparatus. . . .

in suction irrigation, as it runs over the field, tends to blur the field when the operating microscope is used. This occurrence is disturbing, if not a major drawback.

After the uncapping maneuver, I direct a stream of fluid into the fistula, which is then examined under the operating microscope. This gentle washing out is continued until the fistula and the membranous canal are clear. An argument put forward in favor of constant irrigation is the dissipation of heat. An interruption made every few seconds of the grinding maneuver, as Lempert does, and irrigation of the field by hand prevent the generation of heat.

Lister Building, Jeppe and Smal Streets.

MELKERSSON'S SYNDROME

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IN 1928 Melkersson [1] described an unusual syndrome consisting of recurrent peripheral facial paralysis associated with chronic edema of the face and lips. Inasmuch as the cause and the pathologic changes in this puzzling condition remain uncertain, the experience with an additional case is deemed worthy of reporting.

According to Kettel,[2] Melkersson's syndrome is recognized at the present time as a triad of findings: (*a*) peripheral facial paralysis, (*b*) chronic angioneurotic facial edema and (*c*) lingua plicata. The peripheral facial paralysis in this syndrome is indistinguishable from Bell's palsy. The angioneurotic edema is chronic and is most often localized to the lips, usually the upper lip. The deeply furrowed tongue, or lingua plicata, was added to the syndrome by Rosenthal [3] in 1931.

Kettel has pointed out that the disease frequently begins in childhood or youth, in the majority of cases before the eighteenth year. Swelling of the face may occur at the time of the initial facial paralysis, but in many cases several years may elapse before the edema appears. The swelling is localized to the eyelids, nose, chin and, especially, to the lips. In 3 of 5 of Kettel's patients the swelling was confined to the upper lip. At first, the edema is transient, later progressing, after many acute exacerbations, to a permanent chronic swelling, with minor variations in size from time to time. Clinically, the edema resembles angioneurotic edema, but it does not occur elsewhere in the body. The edema is soft, nonpitting, nontender and noninflammatory. The skin is of normal texture, and the exposed mucous membrane is often dry and fissured. There is no regional adenopathy.

The facial paralysis in Melkersson's syndrome is always of the peripheral type; the paralysis may be recurrent, bilateral and familial. The onset of the paralysis is sudden and often complete. Recovery is slow but it is usually without residual weakness.

Read before the Virginia Society of Ophthalmology and Otolaryngology, Richmond, Va., May 1949.

1. Melkersson, E.: Hygiea **90**:737, 1928.

2. Kettel, K.: Melkersson's Syndrome: Report of Five Cases, with Special Reference to Pathologic Observations, Arch. Otolaryng. **46**:341 (Sept.) 1947.

3. Rosenthal, C.: Ztschr. f. d. ges. Neurol. u. Psychiat. **131**:475, 1931.

The anomaly of the tongue appears long before the onset of the facial edema and Bell's palsy and probably is of hereditary nature. There is probably no connection between the facial edema and the furrowed tongue, although cases have been observed in which the tongue showed a swelling simultaneously with the lips.

In 1933 New and Kirch [4] reported 67 cases of chronic noninflammatory permanent enlargement of the face and lips; in 13 of these cases there was associated facial paralysis. A soft, nonpitting, smooth enlargement, usually involving the upper lip, was noted; edema of the cheeks, lower lip, tongue and eyelids was next in order of frequency. The swelling was not related to exposure to cold, trauma, ingestion of food or intercurrent illness. Recurrence of the edema appeared at intervals of from three weeks to seven months, affecting the same site. After repeated attacks, permanent residual swelling was noted. In 1 patient the first attack of facial paralysis occurred at the age of 4 years, and she had five subsequent episodes involving each side of the face. The facial edema started twenty-two years after the initial facial paralysis; in other cases the paralysis started from twenty-five years to six months before the onset of the facial edema.

The cause of the edema is not known. The name angioneurotic edema is often used to indicate a condition of periodic vasodilation and transudation due to various factors, i. e., allergy, endocrine dysfunction, focal infection. Often the edema is of idiopathic origin. The vasodilator element is said to be a histamine-like substance. In angioneurotic edema, however, there is involvement of other parts of the body and the condition is usually transient; "Melkersson's swelling" is confined to the face.

Vaguely, this condition has been thought to be a peculiar disorder of the vasomotor system. Kettel stated that the facial edema is an angioneurotic edema due to a disturbance in the regulation of possible vasomotor fibers in the facial nerve, a neurotrophic facial edema. He expressed the opinion that the facial paralysis is caused by a disorder of vasa nervorum. "The nerve is paralyzed because there is a block of the blood vessels, and as a consequence of this it secondarily becomes edematous . . . and is compressed in the facial canal." There is general agreement that on surgical exposure in the average case of Bell's palsy the nerve is edematous and constricted by the nerve sheath, although the nerve appeared normal in 2 of Kettel's cases of Melkersson's syndrome. The osseous necrosis of the mastoid process and in the wall of the facial canal found by Kettel in some instances is thought to be due to the same process, an ischemic bony necrosis.

4. New, G. B., and Kirch, W. A.: Permanent Enlargement of Lips and Face Secondary to Recurring Swellings and Associated with Facial Paralysis: Clinical Entity, J. A. M. A. 1230 (April 22) 1933.

In a discussion on the nature of Bell's palsy, Hilger [5] emphasized that the compression of the nerve due to edema is usually insufficient to disrupt the axis cylinder and that the paralysis is a result of local vascular changes producing ischemia and interrupted conduction of the motor nerve, sometimes with and sometimes without secondary edema. Hilger posed the question, "Why and by what mechanism are ischemia and edema produced in so localized a region?" He concluded that Bell's paralysis is an ischemic neuritis resulting from segmental arteriolar spasm and seen in those persons with an inherent tendency for focal dysfunctions of the autonomic nervous system.

The facial paralysis, according to Kettel, is a result of decreased function of the motor fibers of the facial nerve, and the facial edema is a result of a disturbance of the innervation of the vasomotor fibers in the same nerve.

The relation between blepharochalasis, Ascher's syndrome and Melkersson's syndrome is not known. Blepharochalasis is a chronic recurring edema of the eyelids, associated at times with so-called double lip or macrocheilia. This condition is said to be some peculiar disorder of the vasomotor system. Ascher's syndrome is blepharochalasis associated with hyperthyroidism and double lip.

There is apparently no classification available of the broad variety of chronic swellings of the various facial structures. Many types of pathologic lesions are reported in the literature, and little is known of the associated abnormalities.

Few articles on Melkersson's syndrome include a pathologic report on tissue excised from the edematous facial areas, and these microscopic observations are not in agreement. In some cases the sections were removed after treatment had been instituted. New and Kirch [4] stated that tissue was taken for biopsy in a few of their cases but that nothing was found other than edematous tissue containing lymphocytes. Conway [6] in 1938 described 3 cases of macrocheilia; in 1 case there was a permanent enlargement of the lips, associated with facial paralysis and furrowed tongue. On biopsy, the enlargement of the lips was found to be due to hyperplasia of the labial mucous glands and diffuse inflammation of the submucous tissue. Kettel mentioned a report by Ekbom and Wahlström on the microscopic examination of 1 case of Melkersson's syndrome. Their report follows:

> The preparation consists of striated muscles and connective tissue with round cell accumulations. The muscular bundles are not, as in normal muscles, placed side by side but are separated by cavities. The latter are found to be empty, when the sections are examined, and they may possibly be dilated lymphatic spaces. Further, under the epithelium a sclerotic connective tissue can be observed containing numerous ectatic lymphatic vessels.

5. Hilger, J. A.: Laryngoscope **59**:228 (March) 1949.

6. Conway, H.: Surg., Gynec. & Obst. **66**:1024 (June) 1938.

In Kettel's cases the pathologic observations are reported in detail. He found "more or less pronounced edema, especially of the subcutis and the muscles, chronic inflammatory infiltrations, slight dilatation of the blood vessels, slight muscular atrophy and, in a few cases, a partially marked proliferation of the intermuscular connective tissue." Dilated lymphatic vessels were infrequent and hyperplastic labial glands were not seen. Edema and chronic inflammatory reaction were the salient features.

REPORT OF A CASE

The patient, a white woman aged 30, was first seen by me in my office on May 3, 1943. She presented at that time peripheral facial paralysis on the right side. No further abnormal physical findings were noted at that time. The paralysis completely subsided in ten weeks.

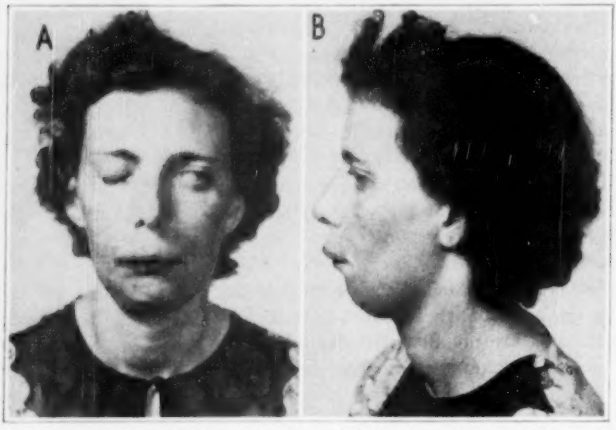

Fig. 1.—*A*, photograph of patient showing left facial paralysis; *B*, classic edema of the upper lip.

She was next seen on July 3, 1948 with a well developed peripheral facial paralysis on the left side. The pronounced edema of the upper lip was first noticed at this time. She stated that her upper lip had been swollen off and on for four years; it first appeared more noticeable on the right side and now, since the onset of the left facial paralysis, it became more pronounced on the left side. The appearance of the upper lip was made more conspicuous by a protrusion of the upper incisor teeth. The patient complained of roaring in and a deep-seated ache around the left ear. No history was obtained of exposure to cold, emotional upset or other precipitating factors.

The results of a general physical and neurologic examination were negative except for the presence of a two months' pregnancy and a deeply furrowed tongue. The examination of the ears revealed nothing abnormal. The hemoglobin was 13 Gm. (84 per cent) and the white cell count was 8,050. The results of the Wassermann test and the urine analysis were negative. A roentgenogram of the skull

showed no departure from the normal. Roentgenographic examination of the chest showed the heart and lungs to be normal. No evidence of angioneurotic edema was found elsewhere in the body. There was no regional adenopathy.

The facial paralysis on the left improved slowly and cleared completely by November 1948. The edema of the upper lip remained unchanged except for small fluctuations in size. Repeated courses with various antihistamine drugs were tried, with no apparent benefit. An allergic survey showed the patient to be moderately sensitive to dust, halibut, tea, chocolate, cocoanut, oranges, grapefruit, prunes, corn

Fig. 2.—Photograph showing lingua plicata.

and spinach. Under allergic management of several months' duration no improvement was noted. The use of calcium, thiamine hydrochloride and vitamin B complex had no effect on the edema of the lip.

On December 4 a plastic procedure was performed on the upper lip. A wedge-shaped section of the upper lip was excised. Recovery was uneventful.

Microscopic examination of the excised tissue was reported by Dr. Thomas Moran, pathologist of the Memorial Hospital:

"The sections of lip show a histologic appearance which is entirely compatible with the clinical diagnosis of Melkersson's disease. Definite changes are seen in all layers of the lip. There is pronounced edema and congestion, especially promi-

nent in the muscle layers, where a large quantity of edema fluid is seen between the individual muscle bundles. Many arterioles in this area show moderate to marked intimal thickening. A definite increase in fibrous tissue is noted in the submucosa, and numerous large and small collections of lymphocytes, plasma cells, and inflammatory exudate appear to be more prominent around the blood vessels. Marked thickening of the epithelium, especially of the rete cones is also noted. In summary, the characteristic changes are edema and congestion of the muscular layer, thickening of the arterioles, fibrosis and chronic inflammation of the submucosa and thickening of the epithelium with edema of the basal cells."

Dr. J. R. Cash, pathologist of the University of Virginia Hospital, also examined the sections from this case. His description of the lesion follows: "Both sections show essentially the same changes. The epidermis is considerably thickened, due to enlargement of the rete cones, which compress the papillae of the corium. In the deeper layers of the epidermis there are small irregular groups of large clear epithelial cells which appear to be edematous. These are quite sharply marked off from the surrounding epidermal cells, which are of normal appearance. There is no special activity of the basal layer. The surface of the skin has lost its granular layer over most of its extent, the stratum granulosum being present in only very small areas, often limited to three or four cells. There is, in general, a thin layer of parakeratosis of the superficial epidermis, which tends in places to desquamate. No inflammatory changes are seen in the epidermis proper. The main site of the lesion lies in the dermis and underlying striated muscle. Throughout this entire extent the connective tissue is increased in amount. This increase of connective tissue consists of thickening of the collagen about all of the blood vessels, arteries, arterioles, veins and venules. Many of the small arteries appear to be contracted and have much thicker walls than normal. There is no change in the intima of any of these vessels, however, and the lumen appears to be rather good in all of them. A number of other spaces lined with a single layer of endothelium, which apparently comprises their entire wall, may be lymphatics. A few of these contain occasional red blood cells. It is therefore impossible to say with certainty which are the lymphatics. The increase of collagen extends throughout the papillae and the entire corium. About the vessels are infiltrations composed mostly of lymphocytes and monocytes. A few polymorphonuclear cells are present. In the deeper layers of the corium there are rather circumscribed areas of edema, in which the loose fibers of connective tissue are pushed apart by coagulated fluid. This fluid contains numerous polymorphonuclear leukocytes. The papillae of the dermis are narrowed, apparently owing to compression of the rete cones of the overlying epidermis. The collagen in the papillae, as well as in all layers of the dermis, is much increased. The upper layers of the dermis show a rather homogenous infiltration by lymphocytes, mononuclear cells and a few plasma cells. In the deeper layers of the corium, which show even more pronounced increase of collagen, the cellular infiltration is largely restricted to the tissue around the blood vessels. Large arteries and veins apparently show less striated muscle beneath the corium, which is also shown on this specimen. Larger muscle bundles are separated by collagen, which infiltrates the bundle and even separates the various fibers. There is a slight infiltration of mononuclear cells and lymphocytes throughout the muscle."

COMMENT

This lesion seems to me to be a chronic inflammatory process of the corium. It is impossible to say whether an original obstruction of lymphatics could have caused this picture. I should be inclined to think

of the reverse process as being true, though one cannot with certainty pick out the lymphatics in this case. It would seem that any lymphatics which might be present would be compressed. No hair follicles, sweat glands or sebaceous glands were seen in this specimen.

The therapeutic management of Melkersson's syndrome is directed toward (1) relief of the facial paralysis and (2) correction of the permanent areas of facial edema. At the onset of the facial paralysis, vasodilating drugs, such as nicotinic acid, histamine or papaverine may be of some value. Since 85 per cent of the patients with facial paralysis will recover spontaneously, surgical decompression of the facial canal according to the Ballance and Duel method is reserved for those persons who fail to show signs of returned function of the nerve after an observation period of two months or in whom spontaneous recovery has come to a standstill before complete regeneration has taken place. Electrical stimulation and massage of the facial musculature aid in the restoration of complete facial function.

The correction of the permanent facial edema is by surgical excision of the redundant tissue, preceded, according to New and Kirch, by injections of boiling water into the swollen tissue and the use of radium. In a recent communication, New stated that he has seen a great many patients with this condition since his report in 1933 and that he continues to take care of them as he did then.

If only excision is done, there is a tendency for a recurrent attack of facial edema, although this method was used with success in one of Kettel's patients.

After the operation, in this case the cosmetic result was satisfactory until there occurred another episode of acute edema, four months later, which produced a slight temporary enlargement of the lip. Roentgen ray therapy has been tried and the appearance of the upper lip at the present time is greatly improved.

SUMMARY

Melkersson's syndrome is applied to the association of recurrent peripheral facial paralysis and chronic angioneurotic edema of the face. Clinical observations and pathologic data on an additional case of Melkersson's syndrome are here reported.

PRESSURE CHANGES AND BAROTRAUMA RESULTING FROM DECOMPRESSION AND RECOMPRESSION IN THE MIDDLE EAR OF MONKEYS

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THERE are three situations in modern life under which persons are exposed to the danger of receiving barotrauma from sudden change in atmospheric pressure: flight at high altitude, working in the caisson or escaping from a submarine and exposure to bomb blast. It is generally agreed that the outstanding pathologic effect of sudden barometric changes is on those organs which contain air. Among those organs the middle ear is evidently a vulnerable one.

Although the otic symptoms incidental to sudden pressure changes have long been described clinically and interpreted in various ways, the hemorrhages in the middle ear cavity and the mastoid cells and, especially, the mechanism involved in the production of these pathologic changes have never been adequately investigated.

Alt [1] was probably the first who described the apoplectiform lesions in the ear of caisson workers and correctly interpreted the tympanic hemorrhage as being due to the inequality of pressures inside and outside the ear drum. Literature on caisson sickness has been steadily increasing since then. So far as the experimental approach to this subject is concerned, there had been a long dormant period until 1929, when Vail [2] reported 2 cases of middle ear bleeding from air compression in a caisson and performed a series of experiments to reproduce the symptoms and lesions in various animals.

From the Aeromedical Research Unit, Department of Physiology, Yale University School of Medicine.

This investigation was supported by funds from United States Navy (Office of Naval Research Contract N6-ori-44 Task Order VII).

1. Alt, F.: Ueber apoplectiforme Labyrintherkrankungen bei Caissonarbeitern, Monatsschr. f. Ohrenh. **30**:324-326, 1896.

2. Vail, H. H.: Traumatic Conditions of the Ear in Workers in an Atmosphere of Compressed Air, Arch. Otolaryng. **10**:113-126 (Aug.) 1929.

The investigators on the subject of experimental air blast concussion, such as Mairet and Durante,[3] Hooker[4] and Zuckerman,[5] were more interested in the pulmonary lesions. They paid little attention to the damage to the ear by bomb blast, the most drastic form of pressure change.

The pioneer work bearing directly on the effect of pressure change on the middle ear seems to be that of Armstrong and Heim[6] in 1937, amplified by Armstrong[7] in 1939. They made a detailed observation on the physiology of the eustachian tube under marked variation of atmospheric pressure. They also introduced the term "aero-otitis media" in describing the effects caused by the drastic change of pressure. Since their observations were limited to human subjects, they did not determine quantitatively the pressure changes in the ear, nor did they observe the pathologic changes which cannot be recognized without acute experimentation and postmortem examination.

A number of studies appeared recently which either stressed the barometric effect on the hearing ability[8] or dealt with the preventive and therapeutic aspects of aerotitis media.[9] The pathology and the mechanism of otitic barotrauma have been experimentally studied on cats by Dickson, McGibbon and Campbell.[10]

In the present investigation we have attempted to measure directly the pressure changes in the middle ear under various conditions; to

3. Mairet, A., and Durante, G.: Étude expérimentale du syndrome commotionnel, Presse méd. **25**:478, 1917; Contribution à l'étude expérimentale de lésions commotionelles, Rev. neurol. **35**:97-110 (Feb.) 1919.

4. Hooker, D. R.: Physiological Effects of Air Concussion, Am. J. Physiol. **67**:219-274 (Jan.) 1924.

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6. Armstrong, H. G., and Heim, J. W.: The Effect of Flight on the Middle Ear, J. A. M. A. **109**:417-421 (Aug. 7) 1937.

7. Armstrong, H. G.: Principles and Practice of Aviation Medicine, ed. 2, Baltimore, William Wood & Company, 1943, chap. 10.

8. Loch, W. E.: Effect of Experimentally Altered Air Pressure in Middle Ear on Hearing Acuity in Man, Ann. Otol., Rhin. & Laryng. **51**:995-1006 (Dec.) 1942. Shilling, C. W., and Everley, I. A.: Auditory Acuity Among Submarine Personnel, U. S. Nav. M. Bull. **40**:664 (July) 1942. Shilling, C. W.: Aero-Otitis Media and Auditory Acuity Loss in Submarine Escape Training, Tr. Am. Acad. Ophth. **49**:97-102, 1944.

9. Fowler, E. P., Jr.: Use of Radon to Prevent Otitis Media Due to Hyperplasia of Lymphoid Tissue and Barotrauma (Aero-Otitis), Arch. Otolaryng. **40**:402-405 (Nov.) 1944; Irradiation of the Eustachian Tube, ibid. **43**:1-11 (Jan.) 1946.

10. Dickson, E. D. D.; McGibbon, J. E. G., and Campbell, A. C. P.: Acute Otitic Barotrauma—Clinical Findings, Mechanisms and Relationship to the Pathological Changes Produced Experimentally in the Middle Ears of Cats by Variations of Pressure, J. Laryng. & Otol. **58**:465-488 (Dec.) 1943.

analyze the process of the development of the positive and negative pressures in the middle ear during decompression and recompression; to study the role played by the eustachian tube in the regulation of the intra-aural pressure; to determine the critical value of middle ear pressure at which the tympanic hemorrhage occurs, and to explore the factors contributary to the production of the hemorrhage in the tympanic cavity and mastoid cells.

METHODS

Twelve macaque monkeys (19 experiments) were used in the present investigation. In most of the experiments animals were under pentobarbital sodium (35 mg. per kilogram) anesthesia. A few unanesthetized monkeys were also used. As a routine procedure the drum membranes were punctured prior to the experiment with the aid of an otoscope in order to make the middle ear cavity and the external meatus communicate with each other. The ear was then connected with the barometric device, which consists of a membrane-mirror manometer made by fastening a taut, unvulcanized rubber membrane (about 1.5 mm. thick) onto the hub of a 17 gage syringe needle. The connection between the manometer and the opening of the external auditory meatus was made by a short, small but very strong rubber tubing adopted from the sheath of rubber-insulated electric wire. Collodion was used to secure the connecting tube to the ear and to make it air tight. The inner diameter of the tube is about 1 mm. and the total dead space in the tube and in the manometer was estimated as no more than 20 cu. mm.

After the manometer was installed in the ear, both the animal and the manometer were fixed in position to the animal holder. They were then put into the pressure chamber. The pressure chamber used in this investigation consists of two compartments. The main compartment is made of a steel cylinder, about 1 meter long and 60 cm. in diameter, which is communicable with the small animal chamber through a porthole and an adjustable valve. The two chambers are partitioned by a paper diaphragm which can be readily burned out electrically when explosive decompression is desired. The air in the main chamber can be evacuated by a vacuum pump, so that any altitude can be simulated by adjusting the degree of evacuation. The detailed description of this pressure chamber has been given by Gelfan, Nims and Livingston.[11] Use of an enlarged plexiglass® animal chamber adapted for monkeys [12] permitted not only direct observation of the animal but also optical recording of the manometer oscillations.

In the recording system, a condensed beam of light after passing through a narrow slit is projected to the small concave mirror on the membrane manometer and then reflected to a kymograph camera. The signals indicating pressure or altitude are marked electromagnetically on the side of the photographic paper.

In the experiments with unanesthetized animals, the head, the shoulder and the upper part of the chest were always immobilized with plaster of paris while the animal was under anesthesia. The experiment would not start until the cast

11. Gelfan, S.; Nims, L. F., and Livingston, R. B.: Explosive Decompression at High Altitude, Yale Aeromedical Research Unit Report no. 56, July 1, 1947.

12. Gelfan, S.: Explosive Decompression of Monkeys to Extreme Altitudes and Recompression at Free Fall Rate, to be published.

became firm and the animal had completely regained consciousness. In spite of the rigid harness of the animal, the records still cannot be made completely free of the undesirable disturbances caused by the struggling movements. These disturbances, however, can be readily distinguished from the displacement of the tracings produced by true pressure changes.

The manometer was calibrated at the end of each experiment. Within the pressure range of + 600 and — 600 mm. of mercury, the relation of pressure and displacement of manometer tracing is perfectly linear.

Oxygen was adjustably supplied during the experiment. The air turbulence in the animal chamber created by the oxygen stream might sometimes cause slight fluctuations of the middle ear pressure, which, however, is negligibly small.

RESULTS

1. *Periodic Changes of Middle Ear Pressure During Decompression.*—Under the conditions existing immediately before the decom-

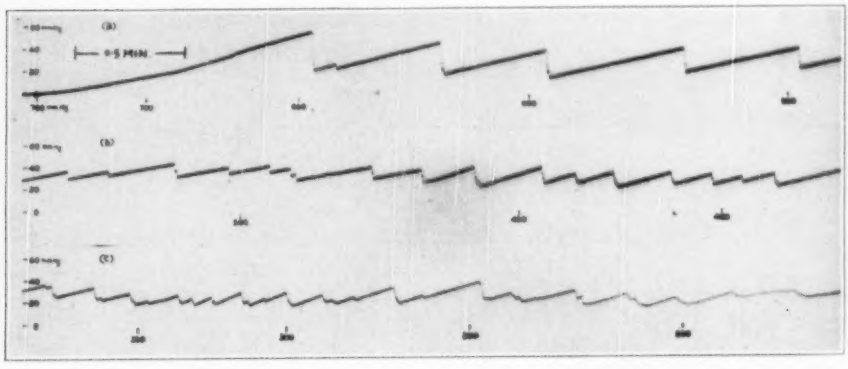

Fig. 1.—Photographic records of pressure changes in the middle ear during decompression (*a*, *b* and *c* are a continuous record). In this and all the other similar pictures, the scale marked with numbers at the beginning of each record indicates the magnitude of tympanic pressure in millimeters of mercury, and the vertical lines marked with numbers beneath the tracings indicate the ambient pressure of the animal during the course of decompression. Macaque 2250L; pentobarbital sodium anesthesia; Jan. 1, 1949.

pression started, the air pressure in the middle ear cavity as well as in the manometer system remained in equillibrium with the ambient pressure at sea level.

At the beginning of decompression the tympanic cavity behaved like a rigid physical system, i. e., the tympanic pressure increased in inverse proportion to the ambient pressure, strictly according to Boyle's law. In other words, the positive pressure in the middle ear increased as the animal was sent higher and higher up to a certain simulated altitude when the positive pressure developed in the ear was high enough to force the eustachian tube open, thereby equalizing the middle ear with

the ambient pressure. The reduction of the positive tympanic pressure automatically led to the closure of the eustachian tube, because the eustachian tube is only a potential tube which is always in a collapsed state unless it is passively distended or opened by the contraction of the tensor veli palatini muscle.

As the animal was further decompressed, the middle ear pressure rose again until the next opening of the eustachian tube was effected. The same sequence of events repeated themselves again and again as the animal was decompressed more and more. The slow rising and the sudden falling of the tympanic pressure in the course of decompression gave a sawtooth appearance to the curve registered on the manometric record (fig. 1).

TABLE 1.—*Maximal Tympanic Pressure Before the Initial Opening of the Eustachian Tube*

Animal	Ambient Pressure, Mm. Hg.	Simulated Altitude		Tympanic Pressure, Mm. Hg.
		Meters	Feet	
2242L	644	1,373	4,500	123
2243L	654	1,250	4,100	90
2250L	661	1,159	3,800	80
2248R	676	976	3,200	68
2252L	669	1,068	3,500	64
2248Lb	676	976	3,200	57
2244R	650	1,310	4,300	54
2141L	709	580	1,900	53
2245Lc*	681	915	3,000	52
2245La*	681	915	3,000	50
2245Ld*	681	915	3,000	50
2251R	725	427	1,400	42
2244L	694	763	2,500	36
2249R	726	381	1,250	36
2249L*	728	366	1,200	34
2281L*	725	396	1,300	32
2246R	699	701	2,700	31
2250Rb*	699	823	2,700	27
2250Ra*	746	152	500	12
Average	690	813	2,666	52 ± 24

* Unanesthetized animal.

2. *Pressure Required for Initial Opening of the Eustachian Tube.*—During the climbing for the first several hundred meters above sea level, the eustachian tube in most cases remained closed; it apparently can withstand a positive pressure within the tympanic cavity amounting to a value of about 52 mm. of mercury. The eustachian tube would give way for the first time when the tympanic pressure rose above that value. The data bearing on this point are presented in table 1. In this table the first column indicates the number of monkeys on which the observations were made. The second and third columns indicate respectively the ambient pressure and the corresponding simulated altitude at which the first opening of the eustachian tube occurred. The fourth column gives the maximum tympanic positive pressure developed immediately before the initial opening of the eustachian tube. As shown in this table the

average value of the tympanic pressure in nineteen observations is 52 mm. of mercury with a standard deviation 24.8. This figure is significantly high in comparison with the pressures needed for the subsequent openings of the eustachian tube. It is believed that in the initial opening of the eustachian tube greater force is necessary to overcome the resistance offered by the accumulated mucus in the lumen and around the ostium of the eustachian tube. With respect to the magnitude of this initial tympanic pressure, there were considerable individual variations from animal to animal. In animal 2242L, the maximum pressure amounted to a value of 123 mm. before the eustachian tube gave way, while in animal 2250Rb a pressure of only 27 mm. or even less (12 mm. as in animal 2250Ra) was enough to push the tube open. The especially high value for some animals was probably due

TABLE 2.—*Residual Pressure Remaining in the Middle Ear at the End of Decompression*

Animal	Residual Pressure in the Tympanum, Mm. Hg.
2242L	72
2243L	50
2281L*	41
2253L	40
2250L	26
2251R	25
2245R	20
2241L	20
2244R	20
2246R	16
2249L*	10
2245L*	5
2249R	0
2250R*	0

* Unanesthetized animal.

to some special anatomic or pathologic conditions of the eustachian tube, such as congenital stenosis, inflammation of the mucous membrane in the throat, inflammation or lymphoid hyperplasia of the eustachian tube or obstruction of the otolaryngeal passage by excessive accumulation of thick mucus.

3. *Residual Pressure in the Tympanum.*—As the expanding air discharges through the eustachian tube, the middle ear pressure is promptly reduced. The equalization between tympanic and ambient pressures during decompression, however, is not as a rule complete. A positive residual pressure was usually observed in the tympanum at the moment that the eustachian tube was reclosed. As is shown in table 2, the residual tympanic pressure in most animals studied was around 20 mm. of mercury. This magnitude of residual pressure may be regarded as representing the force exerted by the walls of the compressible part of membranocartilaginous tube in keeping itself normally closed. As

tested by Perlman,[13] the minimal resistance offered by the eustachian tube of the human subjects during autoinflation or Valsalva's maneuver is of about 20 mm. in the erect posture. It is interesting that his figure, although obtained by a completely different method and in completely different subjects, is, incidentally, identical with ours.

4. *Minimum Excessive Pressure Required for Forcing the Eustachian Tube Open.*—The difference of pressures before and after each discharge of air through the eustachian tube was by no means constant. When the animal was progressively decompressed at a slow rate the differential pressure for the initial discharge was invariably higher than for the subsequent discharges. These subsequent discharge pressures also became gradually smaller during the simulated ascent, as may be seen in figure 1. The apparent difficulty in forcing an initial opening of the eustachian tube is believed to be caused by a possible obstruction of the lumen and the ostium of the eustachian tube by mucus which would be cleared off by the forceful expiration of the escaping air.

5. *Rate of Decompression and the Opening of the Eustachian Tube.*—It must be stated here that the periodic discharge of the expanding air through the eustachian tube can be observed only at a slower rate of decompression. When the animal was decompressed at a rate of about 50 mm. of mercury a minute, the periodic opening of the eustachian tube, as manifested by the sawtooth appearance of the manometer tracing, can be readily reproduced. At a higher rate of decompression, say more than 120 mm. a minute, the individual waves may be fused into a plateau resulting from a sustained patency or an incomplete reclosure of the eustachian tube. Ordinarily, it takes some time for the eustachian tube to complete an individual act of opening and closing, especially when the aperture is narrow. It is obvious that during rapid decompression the eustachian tube will be opened continually when the rate of pressure development in the middle ear exceeds the rate of outflow of air through the tube. The eustachian tube in some animals seems unable to close again very tightly once it is forced open, and so the middle ear pressure will follow the ambient pressure resulting from the continuous leaking of air through the patent tube. This observation agrees with the actual experience of fliers and some decompression experimenters. Whaley [14] reported that he had never given a thought to his ears while he was decompressed in the decompression chamber at a rate of 100,000 feet (approximately 3,300 meters) a minute.

13. Perlman, H. B.: The Eustachian Tube: Abnormal Patency and Normal Physiologic State, Arch. Otolaryng. **30**:212-238 (Aug.) 1939.

14. Whaley, J. B.: Problems of Aviation Medicine Relating to Ear, Nose and Throat, Arch. Otolaryng. **36**:438-442 (Sept.) 1942.

Apparently the usual "clicks" attendant on the opening of the eustachian tube [15] did not occur during such a rapid ascent.

If the eustachian tube functions as a perfect flutter valve, the sum of the effective excessive pressures for each individual opening of the eustachian tube and the residual pressure should be theoretically equal to the remainder of the atmospheric pressure (760 mm. of mercury) minus the pressure to which the animal is decompressed. The experimental values, however, were always below this theoretic value. For instance, in experiment 3 (animal 2251R) in which the animal was decompressed to an altitude of 10,700 meters (35,000 feet), at which the ambient pressure is about 180 mm., the sum of all the individual excessive pressures and the average residual pressure was only 508 mm.—that is, 72 mm. less than it should be (760 — 180 = 580). The lack of promptness in the reclosure of the eustachian tube may account for this loss of pressure. It is also possible that the eustachian tube is never completely closed, a small aperture remaining in the upper part of the lumen.

6. *Explosive Decompression.*—As previously mentioned, the periodic rising and falling of the middle ear pressure can be produced only by slow decompression. When the rate of decompression becomes higher the eustachian tube more or less remains open continuously permitting a sustained escape of air from the tympanic cavity. If the rate of air expansion resulting from decompression equals the rate of the continuous outflow of air through the eustachian tube there will be no pressure change in the ear. If the rate of air expansion exceeds the rate of air elimination, as in the case of very rapid decompression, the middle ear pressure would constantly rise until the end of decompression. This lag in equalization of middle ear and ambient pressures with patent eustachian tube is observed to a marked, although momentary, degree during explosive decompression. At the instant of explosive decompression the pressure in the middle ear rises immediately to a high value, falling very quickly to that of the ambient pressure. Obviously, the small opening of the eustachian tube does not permit a decompression rate equal to that of the animal chamber (about 0.2 second for most experiments).

The maximum pressure developed in the ear during explosive decompression approaches the difference in pressure between the predecompression and postdecompression altitudes. The higher the rate of decompression, the closer is this value to that of the difference in pressure. This relation was demonstrated in experiments 5 (animal

15. Graves, G. O., and Edwards, L. F.: The Eustachian Tube: A Review of Its Descriptive, Microscopic, Topographical and Clinical Anatomy, Arch. Otolaryng. **39**:359-397 (May) 1944.

2245R) and 10 (animal 2243L), in both of which the animals were explosively decompressed from a simulated altitude of 9,150 meters (30,000 feet) to that of 16,800 meters (55,000 feet) but at different rates. The rate of decompression was slower in experiment 5 than in experiment 10. The maximum pressure developed in the middle ear cavity in the former was 66 mm. of mercury, whereas that in the latter was 145 mm., which is closer to the figure of 157 mm., the pressure difference between the two altitudes. After reaching its peak, the relative middle ear pressure usually returned quickly to the original level immediately preceding explosive decompression (experiment 5). However, not infrequently the eustachian tube would shut prematurely before the pressure fell to that level. It has also been observed that the closed eustachian tube would be reopened shortly afterward and when that happened the residual pressure in the ear would become lower than the preexplosion level.

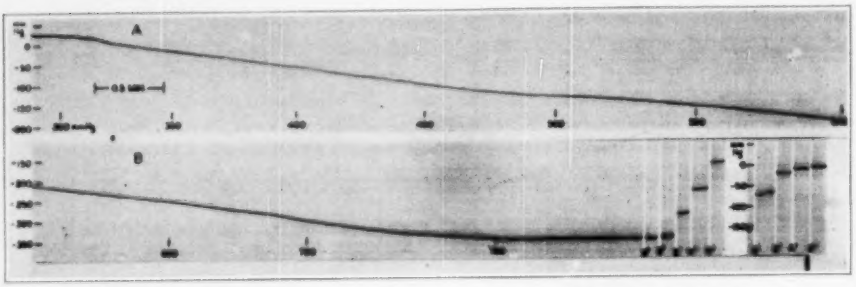

Fig. 2.—Photographic records illustrating the development of negative pressure in the tympanic cavity during recompression from 304 to 760 mm. of mercury (*A* and *B* are a continuous record). Toward the end of record *B* are shown the gradual decrease and final disappearance of the negative tympanic pressure at various intervals after the cessation of recompression at sea level. Macaque 2242R; pentobarbital sodium anesthesia.

7. Genesis of Negative Tympanic Pressure During Recompression.—When anesthetized animals were brought down from simulated high altitudes, the relative middle ear pressure decreased in inverse proportion to the increasing ambient pressure. Because of the flutter-valve-like structure of the eustachian tube, which prevents passive reentry of air into the ear cavity during recompression, the middle ear cavity tends to become like an air-tight closed chamber in which the negative pressure would be completely determined by the ambient pressure. The photographic record in figure 2 shows the uninterrupted development of negative pressure in the middle ear during recompression from a pressure of 300 mm. of mercury to that of 760 mm.

Theoretically, the negative pressure developed in the middle ear during recompression should be equal to the difference between the pressure from which the recompression started and the pressure at which the recompression ended. Consequently, the greater the range of recompression, the higher the negative pressure developed in the ear. For instance, if an animal is sent down from an altitude of 16,800 meters (60 mm. of mercury) to sea level, the negative pressure developed in the middle ear at the end of the recompression should be 691 mm. (760—69). In our experiments, however, the magnitudes of the negative pressure developed was always lower than the theoretic value. Table 3 shows the difference between the theoretic value and the negative pressure actually obtained in different animals. The difference between the theoretic and the practical values of the negative pressure in the ear can be accounted for either by the incomplete closure of the

TABLE 3.—*Relation Between Range of Recompression and Magnitude of Negative Pressure Developed in the Tympanum*

Animal	Range of Recompression			Negative Pressure, Mm. Hg.	
	Pressure, Mm. Hg.	Corresponding Altitude			
		Meters	Feet	Theoretic	Observed
2242L	69-760	18,040	(55,000)-s.l.*	—691	—570
2243L	69-760	18,040	(55,000)-s.l.	—691	—530
2252L	69-760	18,040	(55,000)-s.l.	—691	—689
2241L	148-760	12,800	(39,000)-s.l.	—612	—340
2250R	254-760	8,860	(27,300)-s.l.	—506	—406
2244L	258-760	8,820	(27,000)-s.l.	—502	—382
2242R	304-760	7,610	(23,200)-s.l.	—456	—346
2246R	344-760	6,600	(20,300)-s.l.	—416	—262
2249L	508-760	3,590	(10,700)-s.l.	—252	—171

*s.l. indicates sea level.

eustachian tube, through which some air might leak in, or by the dissociation of gas from the serosanguineous extravasation in the middle ear or by both. It is also highly probable that the swelling of the mucous membrane lining the wall of the tympanic cavity reduces to some extent the space in the middle ear and therefore lessens the magnitude of the negative pressure. The last factor is especially important for the interpretation of the slight, but constant, reduction of negative pressure as shown in animal 2281L.

The relation of the middle ear pressure and the ambient pressure is also illustrated in the curves in figures 3 and 4, which were plotted with the abscissa denoting the time of progressive recompression and the ordinate representing absolute pressure in mm. of mercury and equivalent altitude. The upper curve (broken line) represents the ambient pressure of the animal and the lower one (solid line) the absolute pressure in the middle ear. In figure 3, it is shown that in the process of recompression, as the ambient pressure increases progressively, the

absolute pressure in the middle ear shows only very slight change. Sometimes, especially when the eustachian tube was presumably not perfectly closed, the absolute pressure in the middle ear would progressively increase until it reached the level of the ambient pressure long after the cessation of recompression.

8. *Active Opening of the Eustachian Tube During Recompression in Unanesthetized Animals.*—In anesthetized monkeys the development

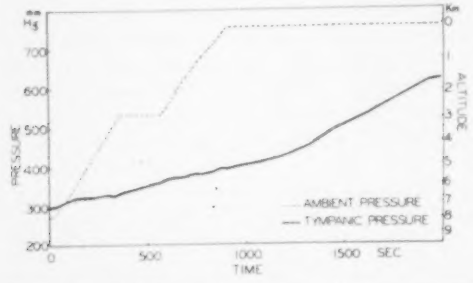

Fig. 3.—The relationship of tympanic pressure and the ambient pressure during and after recompression of an unanesthetized monkey (macaque 2281R). In this figure and all the following similar ones the ordinates represent the magnitude of absolute pressure in millimeters of mercury and the abscissa the time from the beginning of recompression. The difference between the tympanic pressure and the ambient pressure at a given moment of recompression can be regarded as the magnitude of the negative pressure developed in the ear at that moment.

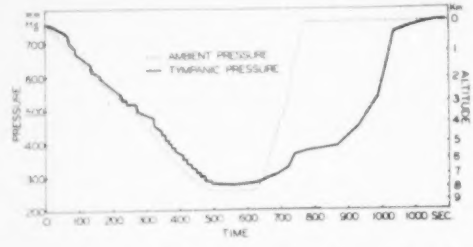

Fig. 4.—Graphic illustration of the relationship of tympanic and ambient pressures during decompression and recompression of a macaque (2244L) under pentobarbital sodium anesthesia.

of negative pressure in the middle ear as previously shown was a continuous process so long as they were progressively recompressed. However, this negative pressure can be equalized readily by active opening of the eustachian tube in some unanesthetized animals. The conscious animals are apparently able to open the eustachian tube as a result of swallowing or other inadvertent actions when the negative

pressure in the ear reaches a certain value. A sample record illustrating this point is given in figure 5 *B*. It is clearly shown in this record that the manometer tracing swings back to the original level three times during recompression, indicating the opening of eustachian tube three times. The relation of the middle ear pressure and the ambient pressure of this animal during decompression and recompression is shown in the curves in figure 6. From the curves in this figure several things are evident. At the beginning of recompression the negative pressure in the middle ear developed up to a value of about —153 mm. of mercury, when the eustachian tube gave way and the middle ear pressure was

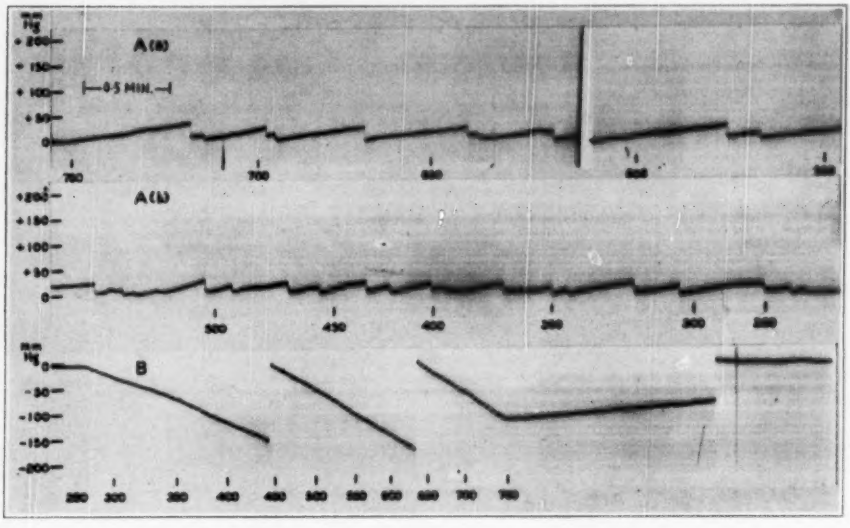

Fig. 5.—Photographic records of the pressure changes in the middle ear of an unanesthetized monkey during decompression and recompression. *A*(*a*) and *A*(*b*) are continuous records during decompression. *B* is the record of tympanic pressure during recompression. Note the equalization of the middle ear pressure twice before and once after the cessation of recompression by the opening of eustachian tube. Macaque 2249R; unanesthetized.

immediately equalized to the ambient pressure. As the recompression was going on, the negative pressure in the ear was reestablished and the same sequence of events repeated itself.

But not all the conscious animals can perform the act of opening the eustachian tube successfully. In one of our experiments (experiment 19) the unanesthetized monkey failed completely to open its eustachian tube and therefore failed to equalize the middle ear pressure, though it could blink its eyes and struggle violently during recompression.

The failure of the unanesthetized animal to open the eustachian tube during recompression is probably due to the fact that once the animal missed the chance to open the eustachian tube at the proper time, when the negative pressure in the middle ear was still low, it would no longer be able to do so subsequently, when the negative pressure in the ear increased beyond a critical value.

The frequency of the active opening of the eustachian tube varies from animal to animal. In some animals, like macaque 2249R (fig. 5), there were only two or three openings of the eustachian tube throughout the whole process of descending from 16,800 meters (55,000 feet) to sea level, while in others, macaque 2250R for instance (fig. 7 *B*), the performance had been repeated twenty-five times in the course of descent from an altitude of 8,400 meters (27,500 feet) to sea level. In the latter case, the negative pressure in the middle ear never exceeded 38 mm. at any time.

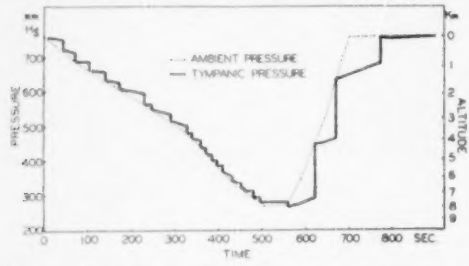

Fig. 6.—Graphic illustration of the relation of the tympanic pressure to the ambient pressure of an unanesthetized animal (macaque 2249R). Note the frequent equalization of the tympanic and ambient pressures during both decompression and recompression.

9. *Rate of Recompression and Functioning of the Eustachian Tube.*—These apparently constitute one of the factors determining whether or not a conscious animal can control the opening of the eustachian tube during recompression. An unanesthetized animal which during recompression at a rate of about 50 mm. a minute (fig. 7 *B*) had been able to equalize the middle ear pressure by frequent and timely opening of the eustachian tube became completely helpless in coping with the rapidly developing negative pressure in the ear while recompressed at a rate of about 200 mm. a minute. Figures 7 and 8 demonstate the effect of rate of recompression on the development of negative pressure in the middle ear. The records in those two figures were taken from the same unanesthetized animal which was experimented on in the same evening and subjected to the same range of recompression. Figure 7 *B* shows the frequent openings of the eustachian tube and the absence of negative

tympanic pressure as a result of slow recompression. Figure 8 *B* shows the development of the negative pressure in the ear and the inability at first and the difficulty later for the animal to open the eustachian tube. It should be noted in these records that the very brief oscillatory excursions of the tracing were caused by the struggling movements of the animal during the experiment. In spite of this kind of undesirable disturbance, the opening of the eustachian tube as characterized by the sudden unidirectional displacements of the tracing can still be clearly seen.

Examination of the records shows no correlation whatsoever between the struggling movements and the openings of the eustachian tube. It

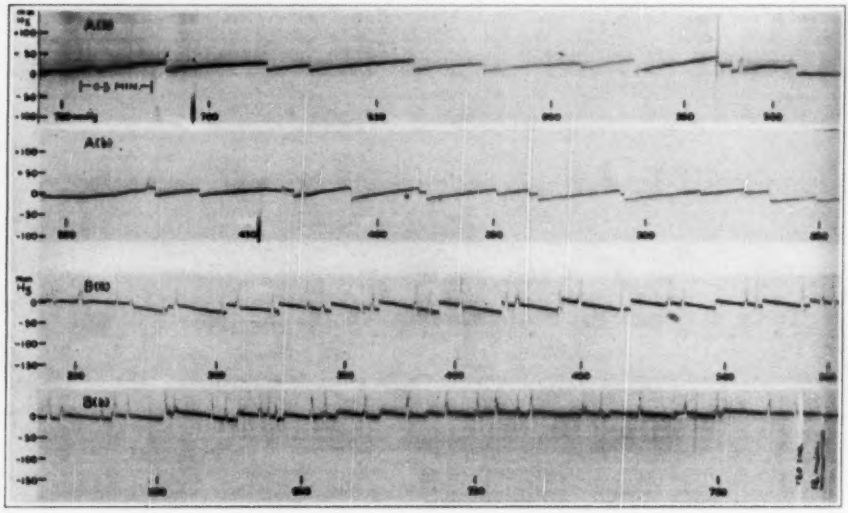

Fig. 7.—Photographic records illustrating the changes of tympanic pressure of an unanesthetized monkey (macaque 2250R) during decompression and recompression. *A*(*a*) and *A*(*b*) are continuous records during decompression, and *B*(*a*) and *B*(*b*), those during recompression. Note the lack of negative pressure in the middle ear during recompression owing to the frequent opening of the eustachian tube. The spikelike oscillations are artefacts caused by the movements of the animal.

is well known from the experiments by Rich [16] that the only muscle which is functionally related to the eustachian tube is the tensor veli palatini, and movements not involving the contraction of this muscle do not influence the patency of the tube.

16. Rich, A. R.: A Physiological Study of the Eustachian Tube and Its Related Muscles, Johns Hopkins Hosp. Bull. **31**:206-214 (June) 1920.

Although the opening of the eustachian tube, whenever it happens, always leads to the same results of equalizing the pressures inside and outside the ear, the mechanism underlying this action during recompression is entirely different from that during decompression. The opening of the eustachian tube during decompression is a passive movement, whereas that during recompression involves an active contraction of the muscle controlling the eustachian tube. As a physiologic process, therefore, the latter performance cannot be achieved in deeply anesthetized animals or in unconscious subjects.

10. *Barotrauma from Recompression.*—Almost all the anesthetized animals which had been decompressed to high altitude showed various

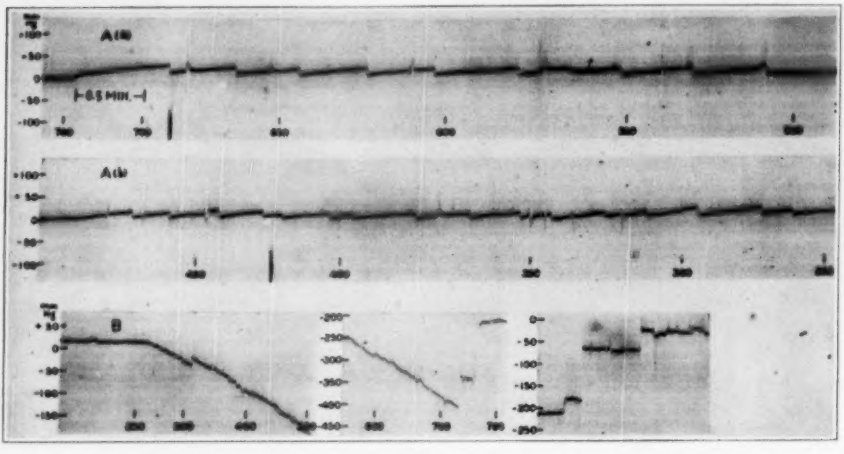

Fig. 8.—Photographic records illustrating the changes of tympanic pressure of unanesthetized monkey (macaque 2250R) during decompression and recompression at a fast rate (200 mm. a minute). *A(a)* and *A(b)* are continuous records during decompression; *B*, during recompression. Note the failure of the animal to equalize the tympanic pressure.

degrees of hemorrhagic lesions in the middle ear. We have never seen any lesions in those monkeys whose drum membranes on both sides had been punctured prior to decompression to simulated altitudes of as high as 75,000 feet (approximately 21,500 meters) and then rapidly recompressed. After unilateral myringotomy prior to the experiment there were no hemorrhages in the middle ear with the perforation in animals decompressed and recompressed. However, figure 9 shows the severe hemorrhage in the mastoid cells of such an animal on the side with

the intact drum membrane. In a previous report from this laboratory [17] hemorrhages were frequently observed through the somewhat transparent tympanic bulla of unanesthetized rats that had been explosively decompressed and recompressed. These hemorrhages were assumed to have occurred in the cochlea. We have repeated these experiments on a few rats and after careful dissection found that the hemorrhages were in the middle ear but not in the cochlea.

The evidence indicates that the tympanic hemorrhage is caused by the negative pressure developed in the middle ear during recompression and not by the decompression, whether the latter is gradual or explosive. While the positive pressure developed in the middle ear after explosive decompression may be high, depending on the final decompression pressure, it is of only momentary duration, since the eustachian tube is

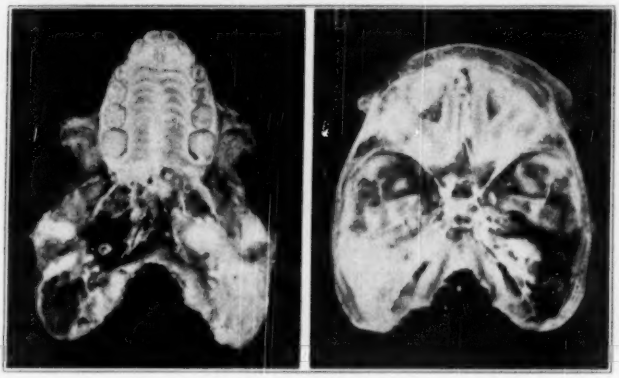

Fig. 9.—Photograph of the skull of a decompressed monkey with the drum membrane on the left side previously punctured, illustrating hemorrhage in the mastoid cells on the right side resulting from recompression (negative tympanic pressure = 675 mm. of mercury); *a*, ventral view; *b*, dorsal view.

forced open. The only effect of perforation of the tympanic membrane is perhaps to increase somewhat the rate of decompression of the middle ear. During gradual decompression of the animal, however, the maximal tympanic pressure before the initial opening of the eustachian tube was almost always below 100 mm. of mercury. Again, this positive pressure is temporary, since it forces the eustachian tube open. The unanesthetized monkeys that succeeded in equalizing the middle ear pressures during recompression had no hemorrhages. The effect of tympanic perforation, therefore, is to prevent the development of negative pressure.

17. Livingston, R. B.; Gelfan, S., and Nims, L. F.: Pathology in Suddenly Decompressed Rats, Federation Proc. **6**:154-155, 1947.

Indirect evidence that positive pressure produces no hemorrhage in the ear is provided by the reports from the laboratories of Wright Field and Randolph Field, in which no mention is made of any ear difficulties following explosive decompression of human being.

It is pertinent here to mention another observation made in this laboratory.[12] Unanesthetized monkeys explosively decompressed to 38,000 to 40,000 feet (12,700 to 13,400 meters) in oxygen do not show any significant changes in heart rate, blood pressure or respiration during a stay of ten minutes or longer at that altitude. During recompression at free fall rate, however, marked bradycardia develops in such monkeys. This effect was interpreted as due to failure to equalize middle ear pressure, since it could be prevented by bilateral myringotomy.

TABLE 4.—*Magnitudes of Negative Pressure and Incidence of Hemorrhage in the Middle Ear*

Experiment	Animal	Rate of Compression, Mm. Hg/Min.	Pressure Developed in Tympanum, Mm. Hg	Tympanic Hemorrhage
11	2252L	165	—689	+++
8	2242L	170	—675	+++
10	2243L	157	—650	+++
19	2250R*	200	—406	+++
14	2244L	192	—382	++
7	2241L	163	—340	+
15	2246R	158	—262	—
21	2249L	44	—171	—
16	2249R*	200	—168	—
18	2245L	630	—160	—
12	2250L	3.6	—138	++

* Unanesthetized animal.

11. *Critical Negative Pressure for Tympanic Hemorrhage.*—Attempts have been made to determine the minimum negative pressure in the middle ear at which the hemorrhage occurs. The data bearing on this problem are presented in table 4. From this table it is evident that the threshold value of negative pressure for the production of the middle ear hemorrhage is probably around 300 mm. of mercury. It must be stated here that there is no sharp boundary between the effective and the ineffective negative pressures. While it may be stated with certainty that negative pressures greater than 300 mm. would cause observable hemorrhage in the ear, a pressure of —168 mm. of mercury, as in the case of macaque 2249R, was also injurious. Traces of extravasated blood were definitely present in the mastoid cells in this animal, although there was no perceptible sign of bleeding on gross examination. It is probable that the negative pressure of such magnitude was not strong enough to inflict a major trauma but was sufficient to cause some slight submucosal bleeding. An exception should also be noted in this table

in the case of monkey 2250L. This animal had a moderate tympanic hemorrhage in spite of the low negative pressure developed in the ear (only —138 mm. of mercury, a figure far below the threshold value). It should be remarked here that the animal in this particular experiment had been recompressed at a very slow rate: It was sent down from a simulated altitude of 12,700 meters (38,000 feet) to sea level in two and a half hours (3.6 mm. a minute) while the rate of recompression on the others was about 160 mm. a minute.

The persistence of negative pressure in the middle ear is obviously a very important factor for the production of tympanic lesions. A low negative pressure in the ear, which does no harm ordinarily, would cause hemorrhage just as effectively as the high negative pressure if it were allowed to persist for a long time.

At face value, a negative pressure of 300 mm. of mercury may not seem high enough to cause hemorrhage in the ear. However, during recompression the walls of the blood vessels in the middle ear are acted on by two pressure forces, the intravascular blood pressure and the negative pressure in the middle ear cavity, both of which tend to distend the blood vessels. The result of these two forces is to increase the strain on the wall of the blood vessels in one direction. However, the precipitating cause for the rupture of the already highly strained blood vessels must be attributed to the "cupping" effect of the negative pressure, which tears the mucous membrane from the bony wall of the tympanic cavity. It is believed that the total strain exerted from both within and without on the blood vessels during recompression is much higher than that exerted unilaterally on the tympanic membrane. This seems to provide a partial explanation as to why hemorrhages in the middle ear often occur during recompression while the drum membrane suffers no rupture.

12. *Gross Pathologic Picture.*—The gross pathologic picture of this kind of lesion involved the bleeding in the tympanic cavity, the tympanic antrum and the mastoid cells of the temporal bone. The extravasated blood in those spaces could be easily seen from the inner and outer surfaces of the temporal bone after it was stripped of periosteal membranes. The dark blood clot was also visible through the tympanic membrane, which itself was always left intact. (These observations were made, of course, on ears to which membrane manometers were not attached.) Careful dissection and inspection of the ear structure of decompressed monkeys revealed no hemorrhage in the cochlea or vestibular organs.

The most striking feature of the severe barotrauma is the profuse bleeding in the mastoid cells of the temporal bone. As is known, the mastoid portion of the temporal bone is hollowed out into a number of irregular spaces containing air. These air-containing mastoid cells

communicate with the tympanic antrum, which, in turn, is continuous with the middle ear cavity. It is also known that the mucous membrane lining the mastoid cells as well as those lining the middle ear cavity are highly vascularized. As we have previously mentioned, during recompression the "cupping" effect of the negative pressure in the middle ear tears the mucous membrane from the bony wall of the tympanic cavity and breaks the submucosal vessels. During recompression the extravasated blood must be sucked into the mastoid cells in which the air had been partially evacuated following decompression. The mastoid apoplexy, because of its recessive location, cannot be detected superficially unless the mastoid bone is exposed, while the hemorrhage in the tympanic cavity can be readily observed through the tympanic membrane by means of an otoscope. However, the mastoid apoplexy is not always accompanied with tympanic hemorrhage. In moderate cases of recompression there may be no perceptible hemorrhage in the middle ear cavity. Nevertheless, the rupture of the submucosal vessels, which would cause serosanguineous deposition in the mastoid cells, is invariably present.

COMMENT

Although the function of the eustachian tube as a regulatory organ for maintaining the equilibrium of pressure inside and outside the ear became common knowledge after Toynbee (1853) elucidated for the first time the physiologic action of the tube, the pressure change in the middle ear under various conditions is recognized only through indirect deductions, namely, from the subjective feeling of fulness or pain in the ear and from the displacement of the tympanic membrane as seen by means of an otoscope or experienced by the subject as sound clicks.

A direct measurement of the pressure change in the ear has never been successfully performed. Politzer [18] was probably the first who attempted to record the pressure change in the tympanic cavity of animals and man by using an instrument named the tympanomanometer, which consists essentially of a bent glass tube partially filled with a colored fluid and fitted to the external meatus at one end. It was assumed that pressure changes in the tympanum may be ascertained by the to-and-fro movements of the tympanic membrane. He described the rising of the fluid level in the manometer during swallowing, which according to him, represents the increase of tympanic pressure caused by the inflow of air through the eustachian tube. After many failures in repeating Politzer's experiments, Jago [19] commented that his manometer was subject to too

18. Politzer, A.: Ueber eine Beziehung des Trigeminus zur eustachischen Ohrtrompete, Wurtz. naturwiss. Ztschr. **2**:92-96, 1861.

19. Jago, J.: The Eustachian Tube: When and How Is It Opened? J. Anat. Physiol. **3**:341-348, 1868.

many sources of errors to be productive of any reliable information. The main defect of Politzer's method, we think, is that the manometer cannot give a quantitative measurement of the intratympanic pressure, since the displacement of the tympanic membrane which Politzer's manometer could show is not a faithful index of pressure change in the tympanum.

The manometer used in the present investigation was designed to measure the pressure changes in the ear directly. This was made possible by increasing the sensitivity of the measuring and recording system and by minimizing the air space in the manometer system and the external meatus which might introduce some influence on the accuracy of the measurements. Because the communication between the tympanic cavity and the external meatus is a prerequisite for success in use of our method, we have confined ourselves to the study only on animals. Nevertheless, it is very feasible to use the same technic to study the human subjects with a perforated drum membrane or on occasions when the myringotomy has been made for some other purposes.

Information acquired from the present findings on the monkeys seems to provide a good quantitative substantiation of the common convictions prevailing in the field of otology. The numerical values obtained from animal experimentation may not be the same as those obtained from the human subjects, but the difference must be very small because of the structural similarity of ears in these two species.

As a preliminary study, a number of factors contributary to the development of negative pressure and the production of barotraumatic lesions in the tympanum and the mastoid bone have not yet been determined. Among these factors the more notable are the influences on the development of tympanic pressure of the blood pressure, the intracranial pressure, the position of the head, the composition of the blood—modified, for instance, by drinking of alcohol—and the composition of the inspired air. The last factor among those mentioned is especially interesting.

Behnke [20] described cases in which serosanguineous filling in the middle ear was found in the subjects during sleep after their return from high altitude while breathing pure oxygen. He pointed out that the delayed barotraumatic lesions in these cases were due to the absorption of oxygen by the capillaries in the tympanic mucosa. According to him, the partial pressure of oxygen left in the tympanic cavity of those subjects was in excess of that in the capillaries, with the result that by the transport of oxygen into the blood a negative pressure was created in the ear. This observation and the interpretation of it are very interesting indeed. However, the results from our experiment would lead us to

20. Behnke, A. R.: Physiologic Effect of Pressure Changes with Reference to Otolaryngology, Arch. Otolaryng. **42**:110-116 (Aug.) 1945.

look at the problem somewhat differently. The negative pressure developed in the middle ear of the anesthetized monkeys during recompression was always gradually reduced with the elapse of time after completion of recompression, regardless of the percentage of oxygen in the chamber in which the animal was decompressed and recompressed. It seems likely that in our experiments the oxygen absorption in the middle ear, if any, could not alter the total volume in the tympanum sufficiently to exceed the change in volume caused by edema and serosangineous effusion. We are inclined to believe that the delayed barotraumatic lesions as reported by Behnke were caused mainly by the prolonged action of a persistent negative pressure of comparatively small magnitude in the ear rather than by absorption of oxygen. As shown in one of our experiments, a negative pressure of 138 mm. of mercury, which would ordinarily cause no perceptible damage to the ear of the animal, produced moderate hemorrhage in the tympanic cavity and mastoid cells when it was allowed to persist in the ear over a long period. The duration of the action of the negative pressure is evidently a very important factor for the production of aerotitis media. Clinical experience has shown that negative intratympanic pressures occasionally developed in some inexperienced flier or plane passengers, unless promptly relieved before passing through the stage of edema, causes exudation and hemorrhage.

The most effective way to prevent the development of negative pressure in the middle ear as well as to relieve the pain therefrom is unquestionably the puncturing of the drum membranes. It has been shown by Canfield and Bateman [21] that pain in the ear from the reduced intratympanic pressure in man could be relieved immediately by myringopuncture without sequelae before the development of later pathologic changes following delayed readjusting of the tympanic pressure. In a report on his experience with the otologic problems in World War II, Canfield [22] urged the recognition of the value of this simple surgical procedure, although he minimized its usefulness in military aviation, because air crews were very well trained in matters of pressure adjustment.

SUMMARY

The pressure changes in the middle ear of macaque monkeys during decompression and recompression have been directly measured by means of a membrane-mirror manometer and recorded photographically.

21. Canfield, N., and Bateman, G. H.: Myringopuncture for Reduced Intratympanic Pressure, J. Aviation Med. **15**:340-343 (Oct.) 1944.

22. Canfield, N.: Otolaryngology in the European Theater of World War II, Ann. Otol., Rhin. & Laryng. **55**:108-120 (March) 1946.

In the course of decompression at a slow rate (50 mm. of mercury a minute) the eustachian tube opened periodically to keep the tympanic pressure always close to the ambient pressure.

The pressure required for the initial opening of the eustachian tube (about 52 mm. of mercury) was higher than that required for the subsequent openings, which seemed to become gradually smaller toward the later stage of decompression.

In most cases a residual pressure of 20 mm. of mercury was present in the tympanic cavity despite the frequent openings of the eustachian tube.

The periodic opening of the eustachian tube occurred only when the decompression rate was low. When the rate of decompression was higher than 120 mm. of mercury a minute, a sustained patency of the eustachian tube would result.

At the instant of explosive decompression the middle ear pressure increased abruptly to a value which tended to approach the ambient pressure difference between the predecompression and the postexplosive decompression pressure. This excess middle ear pressure very quickly returned to that of the ambient pressure.

During recompression, anesthetized monkeys could not equalize the tympanic and ambient pressures and a negative pressure developed in the middle ear, the magnitude of which is determined by the range of recompression.

Unanesthetized monkeys were able to equalize the tympanic and ambient pressures by active opening of the eustachian tube, which, however, could not be performed when they were recompressed at a very fast rate.

When the negative pressure developed up to a value of about 300 mm., severe mastoid apoplexy and tympanic hemorrhage would occur. A negative tympanic pressure of lower magnitude could produce only submucosal hemorrhage. However, the prolonged action of a negative pressure of 138 mm. of mercury could lead to the same degree of severe damage to the ear as does the high negative pressure.

Myringopuncture can prevent the development of negative pressure and therefore prevent the production of barotraumatic lesions to the ear.

EPIDERMOID TUMORS OF THE FRONTAL BONE, SINUS AND ORBIT

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SLOW-GROWING tumors involving the frontal bone, sinus and adjacent areas are not always easily differentiated. This treatise will discuss the rather rare epidermoid type of tumor, with report of a case, and will briefly differentiate other growths with which it may be confused.

An epidermoid tumor, first described in the literature by Cruveilhier [1] in 1829, was termed "pearly tumor" because of its highly refractive and nodular surface. Müller [2] introduced the name "cholesteatoma" in 1838 because of the cholesterol crystals in the mass. Remak [3] originated the hypothesis that such growths arose from embryonic epidermal rests. Heimendinger,[4] Cushing,[5] Skillern,[6] McFarland,[7] Ewing,[8] Coates,[9]

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1. Cruveilhier, J.: Anatomie pathologique der corps humaine, Paris, J. B. Bailliere, 1829, vol. 1, p. 341; cited by Rand and Reeves.[17]

2. Müller, J.: Ueber den feinern Bau und die Formen der krankhaften Geschwülste, Berlin, G. Reimer, 1838, vol. 1, p. 50.

3. Remak, R.: Ein Beitrag zur Entwickelungsgeschichte der krebshaften Geschwülste, Deutsches Arch. f. klin. med. **6**:170, 1854.

4. Heimendinger, A.: Beiträge zur pathologischen Anatomie der Kieferhöhle, Arch. f. Laryngol. u. Rhinol. **19**:382, 1907.

5. Cushing, H.: A Large Epidermal Cholesteatoma of the Parieto-Temporal Region Deforming the Left Hemisphere Without Cerebral Symptoms, Surg., Gynec. & Obst. **34**:557, 1922.

6. Skillern, R. H.: Accessory Sinuses of the Nose, Philadelphia, J. B. Lippincott Co., ed. 4, 1923, pp. 53, 136 and 137.

7. McFarland, J.: Surgical Pathology, Philadelphia, P. Blakiston's Son & Company, 1924.

8. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, ed. 3, Philadelphia, W. B. Saunders Company, 1928.

9. Coates, G. M.: Cholesteatoma of the Frontal Sinus, Arch. Otolaryng. **26**:29 (July) 1937.

Critchley and Ferguson,[10] Love and Kernohan,[11] and Bailey [12] have concurred in the belief that primary cholesteatomas or epidermoids are congenital new growths coming from misplaced or aberrant epithelial tissue. Bucy [13] stated that such growths may arise beneath the scalp, within the diploe, between the bone and dura mater, beneath the arachnoid membrane and particularly about the base of the brain or within the ventricle. He stated the opinion that these growths were probably remnants of incomplete separation of the neural from the surface ectoderm and that if these remnants were deposited between two ectodermal structures, such as the brain and the skin, epidural, intradiploic and epicranial tumors were formed. Ewing [14] stated that many epidermoids are of traumatic origin. A history of injury antedating the tumor was noted in several of the cases reported. Mahoney [15] found in the literature up to 1936 reports on only 23 diploic epidermoids out of 142 epidermoids.

PATHOLOGY

The term cholesteatoma has been used in the past to designate a tumor containing a high concentration of cholesterol. Such growths may be primary or secondary. Physicians in our specialty usually think of cholesteatoma or pseudocholesteatoma as the ingrowth of squamous epithelium from the external auditory canal through a marginal perforation of the tympanic membrane in chronic suppurative otitis media, resulting in a mass of desquamated debris high in cholesterol content. These tumors may occur also in the paranasal sinuses, and they have been designated as secondary or as pseudocholesteatoma because they occur after a chronic inflammatory process. The terms primary cholesteatoma and epidermoid are rather confusing unless one understands the actual formation of these growths. As has been pointed out by Cushing,[5] McFarland [7] and Kopetsky,[16] primary cholesteatoma or epidermoids may also arise in the temporal bone and the middle ear.

10. Critchley, M., and Ferguson, F. R.: The Cerebrospinal Epidermoids (Cholesteatomata), Brain **51**:334, 1938.

11. Love, J. G., and Kernohan, J. W.: Dermoid and Epidermoid Tumors (Cholesteatomas) of the Central Nervous System, J. A. M. A. **107**:1876 (Dec. 5) 1936.

12. Bailey, P.: Intracranial Tumors, Springfield, Ill., Charles C Thomas, Publisher, 1933, p. 349.

13. Bucy, P. C.: Intradiploic Epidermoid (Cholesteatoma) of the Skull, Arch. Surg. **31**:190 (Aug.) 1935.

14. Ewing,[8] ed. 4, 1940, p. 1051.

15. Mahoney, W.: Die Epidermoide des Zentralnervensystems, Ztschr. f. d. ges. Neurol. u. Psychiat. **155**:416, 1936.

16. Kopetsky, S. J.: Cholesteatoma, in The 1933 Year Book of the Eye, Ear, Nose and Throat, Chicago, The Year Book Publishers, Inc., 1933, p. 338.

Several investigators stated that both epidermoids and dermoids are the result of fetal inclusion of epidermal cells, which, depending on the depth of the layer or the embryonic age, produce either one or the other tumor. Thus, it seems logical to believe, as suggested by Critchley and Ferguson,[10] Love and Kernohan,[11] and Rand and Reeves,[17] that if only the squamous epithelium is included in the embryonal inclusion an epidermoid arises. However, if the fetal inclusion contains ectodermal epithelium together with mesodermal corium, with its connective tissue, fat cells, sebaceous glands and hair follicles, a dermoid cyst may arise.

Grossly, an epidermoid has a white, glistening capsule, which is frequently described as having a mother-of-pearl appearance. This capsule is frequently very thin and friable and may be difficult to remove from the dura, as pointed out by Bucy.[13] The contents may vary from a firm, more or less caseous, whitish yellow laminated mass, with a soapy or waxy appearance and absence of vascularity, as described by Ewing,[8] Bucy [13] and Rand and Reeves,[17] to that of a softer, whitish or grayish necrotic mass, as mentioned by Skillern [6] and Coates.[9] The appearance depends on the stage of degeneration.

Bailey [12] described the microscopic sections as showing an outside fibrous layer, within which are flattened cells which may contain keratohyalin; the internal portion is composed of degenerated epithelial cells and may show fat and cholesterol crystals. Love and Kernohan [11] stated that the outer or squamous and basal epithelial lining can be identified by keratohyalin granules, which are present only in the superficial layer of squamous epithelium. They said that cornification characteristics are present and that these desquamated cells constitute the contents of the cyst. Bucy [13] found that the lining of the cyst consisted of three layers composed of (1) cornified epithelial cells, which fill the interior of the cyst; (2) a layer of stratified squamous epithelium, and (3) relatively acellular connective tissue. Rand and Reeves [17] described four layers: (1) external or stratum durum—the part corresponding to the pearly sheen containing collagen fibers with little or no trace of cellular formation; (2) stratum granulosum, or cellular layer, containing stratified or cuboid cells—corresponding to the dermis and containing cytoplasm and keratohyalin granules; (3) stratum fibrosum, and (4) stratum cellulosum—the innermost wavy homogeneous laminas with staining properties of keratohyalin.

It becomes apparent that the microscopic picture has been fairly well established. The term primary cholesteatoma does not adequately

17. Rand, C. W., and Reeves, D. L.: Dermoid and Epidermoid Tumors (Cholesteatomas) of the Central Nervous System: Report of Twenty-Three Cases, Arch. Surg. **46**:350 (March) 1943.

describe the pathologic picture of these tumors. Actually, the name cholesteatoma was intended to indicate high cholesterol content, which may or may not be present in epidermoid growths.

REVIEW OF REPORTED CASES

Haeggström [18] in 1916 described a case of cholesteatoma of the frontal sinus which produced orbital proptosis. The globe returned to normal with disappearance of the diplopia after operation. Spencer [19] reported a case of a slow-growing tumor of twenty years' duration, in which the orbit and the right anterior paranasal sinuses were involved. Orbital proptosis and erosion of the bone had occurred. A primary cholesteatoma was found. Eight years after operation, it was necessary to enucleate the eye. Tissue study at that time revealed a squamous cell carcinoma of the orbit and sinuses. Micháil [20] presented a case designated as a primary cholesteatoma of the orbit with proptosis; the tumor had been present for eight years in a 9 year old child. Bone destruction had occurred at the upper outer angle of the orbit. The wound healed slowly after operation. Kahler [21] discussed a primary cholesteatoma of the frontal bone, which resulted in protrusion of the eyeball, decreased vision and headache. The tumor was felt at the superior orbital margin, and roentgenograms revealed a zone of rarefaction. At operation, a cystic tumor below the periosteum was found containing fluid and a cholesteatomatous mass. Bucy [12] reported 3 cases of intradiploic epidermoids of his own and included these in the table of intradiploic epidermoids found in the literature. The tumors of 7 among 16 patients occurred in the frontal region. Two of the 7 patients had headaches and 1 had convulsions. Four patients had no symptoms and were concerned only with the swelling. Roentgenographic findings showed the characteristic defect. Rand and Reeves [17] reported case histories of 11 patients with diploic or cranial epidermoids, which were considered cholesteatomas. The frontal bone was involved much more frequently than the remainder of the skull. They stressed that the roentgenographic appearance is diagnostic. Among the 15 cases of congenital epithelial tumors presented by Love and Kernohan [11] 3 were extradural. One patient aged 43 gave a history of injury at the age of 10. A cystic tumor with bone destruction was found in the parietal region. Rand and Reeves emphasized that extradural epidermoids are characterized by rather smooth erosion of

18. Haeggström, A. A.: Cholesteatoma Removed from the Frontal Sinus, Hygiea **78**:1122, 1916; cited by Spencer.[19]

19. Spencer, F. R.: Primary Cholesteatoma of the Sinus and Orbit: Report of Case Followed by Carcinoma and Death, Arch. Otolaryng. **12**:44 (July) 1930.

20. Micháil, D.: Cholestèatome de l'orbite, Arch. d' opht. **48**:743, 1931.

21. Kahler, O.: Zur Kenntnis der primären Cholesteatome des Stirnbeins, Ztschr. f. Laryng., Rhin., Otol. **23**:227, 1932.

the calvaria and ballooning of the skull tables, with later destruction of one or both tables. Coates [9] described a case which he termed a true cholesteatoma of the frontal sinus, with loss of the sinus floor and most of the external wall and sequestration of the orbital ridge. King [22] presented 8 cases of epidermoids, 4 of which were of the diploic type. Panneton and Roux,[23] Constans [24] and Thornhill and Anderson [25] each reported a case of cholesteatoma of the orbit with characteristic bony changes and exophthalmos. Pincus [26] presented 2 cases in which the orbit was involved, and Kreibig [27] presented 1 case in which the frontal sinus was affected, the involvement being attributed to secondary cholesteatoma since some evidence of chronic inflammatory changes were present. Otrich [28] stated that a foul odor and a milky discharge were found in a case that he considered a primary cholesteatoma of the ethmoid and maxillary sinus.

REPORT OF A CASE

A case of epidermoid tumor of the left frontal bone, sinus and orbit, which I am adding to the relatively few reported in the literature, brings out the salient features that should be emphasized regarding this type of neoplasm.

The patient, male, aged 26, was first seen in November 1942. His general health was excellent, and there was no past history of chronic nasal congestion, sinusitis or recurrent infection of the upper respiratory tract. His only complaint was a markedly protruding left eye, which, he stated, gradually had become pronounced from childhood on (fig. 1). There was a history of injury at the age of 5 over the left brow and frontal region that had required sutures. The patient rarely had headaches, and when they did occur they were mild and associated with excessive use of the eyes. For many years vision in his left eye had not been as good as that in the right. General physical examination, including a neurologic check-up and laboratory tests, indicated nothing abnormal except the local findings about the left side of the forehead and eye. This eye was marked by extreme proptosis and displaced downward. Upward movement of the globe was limited. A firm tumor was found above and apparently behind the left globe. A smaller tumor, 2 cm. in diameter and 3 cm. above the orbital margin, was present in the

22. King, J. E.: Extradural Diploic and Intradural Epidermoid Tumors (Cholesteatoma), Ann. Surg. **109**:649, 1939.

23. Panneton, P., and Roux, R.: Un cas très rare de cholestéatome de l'orbite, Union méd. du Canada **70**:812, 1941.

24. Constans, G. M.: Cholesteatoma of Orbit, Arch. Ophth. **30**:236 (Aug.) 1943.

25. Thornhill, E. H., and Anderson, B.: Extradural Diploic Epidermoids Producing Unilateral Exophthalmos, Am. J. Ophth. **27**:477, 1944.

26. Pincus, F.: Ueber "Cholesteatom" der Orbita, Klin. Monatsbl. f. Augenh. **90**:145, 1933.

27. Kreibig, W.: Zur Kenntnis seltener Orbitaltumoren, Ztschr. f. Augenh. **95**:113, 1938.

28. Otrich, G. C.: Cholesteatoma Involving the Ethmoidal Cells and the Antrum of Highmore, Illinois M. J. **48**:397, 1925.

frontoparietal region. Visual acuity was 20/20 in the right eye; in the left it was limited to counting fingers at 8 feet (3.4 meters). The fundus was normal. The cornea and conjunctiva were normal, even though the lids barely covered the globe. Trans-illumination of the left frontal sinus was impossible because of the extreme proptosis. Roentgenograms showed that the roof of the left orbit was almost gone and that there was a round bony defect at the outer upper angle about 3 cm. above the orbital margin (figs. 2 and 3). The left frontal sinus was opaque.

On November 10 an exploratory Lynch type incision was made for biopsy. No attempt was made at this time to remove the round tumor in the frontoparietal region. The orbital ridge was irregular. A large laminated (onion-layered), cream-cheese-colored mass filled the frontal sinus and extended above and behind the globe. Eighty grams of this material was removed. The floor and posterior wall of the frontal sinus, as well as an area on the lateral wall of the orbit, had been eroded with no evidence of bone remaining in these areas. The mass was lying in direct contact with the dura mater. The ethmoid region was not involved. A large frontonasal opening was made for drainage of any secretions that might accumulate following operation. A diagnosis was made of primary cholesteatoma

Fig. 1.—*A*, photograph of patient taken at age 12, showing some downward displacement and proptosis of the left eyeball from epidermoid tumor of the frontal bone, sinus and orbit; *B*, at age 18, showing further downward displacement and proptosis of the left eyeball. The slight tumor formation in the frontoparietal region is indicated by arrow; *C*, at age 25, showing pronounced downward displacement and proptosis of the left globe and prominent tumor above the orbital ridge in the frontoparietal region from the tumor. Visual acuity in the left eye was limited to counting fingers at 8 feet.

or epidermoid. Microscopic section presented an almost acellular homogeneous mass, with a few degenerated epithelial cells and a few cholesterol crystals. The lining of the cyst consisted of an outer layer of connective tissue, and an inner layer containing keratohyalin and squamous epithelial cells.

On December 3 visual acuity was 20/40 in the left eye. Refraction with the use of a cycloplegic one month later showed an error of + 2.00 sph. for the right eye and + 3.75 sph. ⊃ + 0.25 cyl., axis 180 for the left eye. With a correction of + 0.75 sph. for the right eye and + 2.25 sph. ⊃ + 0.25 cyl., axis 180 for the left eye, vision was 20/20. Later, some swelling developed at the outer margin of the left upper lid with some tenderness.

On March 4, 1943 an incision was made along the eyebrow to its outer edge; after this, the incision was extended vertically to the hair line. The skin flaps

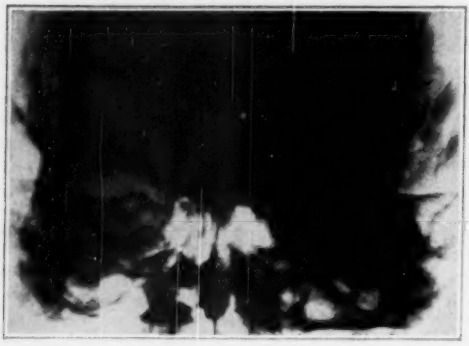

Fig. 2.—Roentgenogram showing front view of skull. Arrow indicates the smooth, round bone defect in the frontoparietal region and opacity of the left orbit caused by the tumor.

Fig. 3.—Roentgenogram of lateral view of skull; arrow indicates the smooth round bone defect in the frontoparietal region.

and periosteum were reflected and the globe was retracted forward and downward. The epidermoidal tract which had eroded the bone was followed up to the area of the round tumor higher on the skull. The outer table had disappeared, and the thin inner table had a hole in the center of the depression through which the dura could be seen. The tumor wall was removed and the adjacent wall along the tract excised so that the dura could be carefully examined. There was no evidence of osteomyelitis. The temporal muscle was reflected and the area was inspected where the tumor had eroded the lateral wall of the orbit. Behind the globe, there was no evidence of recurrence of the mass.

It was necessary to take off about one third of the lateral portion of the orbital ridge because of its irregularity. It was suspected that there was a cyst beneath the dura. This area was explored with a no. 20 needle, but no fluid was aspirated. An attempt to remove the small pearly flakes which were adherent to the dura was partially successful. The surgical field was thoroughly irrigated

Fig. 4.—*A*, recent photograph (front view). The left eyeball has returned practically to its normal position after surgical removal of the epidermoid tumor from the frontal bone, sinus and orbit. Vision was corrected to 20/20 with glasses; *B*, recent photograph (profile view) showing absence of proptosis.

with isotonic sodium chloride solution; ½ inch (0.5 cm.) iodoform gauze pack was inserted into the wound, and the end brought out through a small hard rubber tube placed in an artificially enlarged nasofrontal opening. The periosteum, subcutaneous tissue and skin were closed. The tube and the packing were gradually removed in five days. Irrigations were done several times through the nasofrontal opening.

Some swelling and fluctuation of the lateral portion of the upper eyelid developed in February 1944. A small incision was made, which liberated a small amount of debris. The wound healed and the patient had no further trouble until January 1945, when he returned with fistula in the old incision at the outer angle of the fronto-orbital region. Small scaly white glistening pieces of detritus were irrigated from the area. Penicillin irrigations and instillations were made at three day intervals, and an excision of the tract was done on April 10. Three months later in the same area an opening occurred through which more of these small soft glistening flecks were extruded.

The fistula healed for eight months, after which it broke down and continued to expel more small particles. Even though repeated roentgenograms failed to show any evidence of osteomyelitis, the increase in size of the bone defect indicated bone absorption and the continued presence of epidermoid tissue. Sulfathiazole and sulfadiazine were given until the patient acquired a sensitivity to these drugs. The small opening persisted in spite of penicillin therapy. The patient's temperature has always been normal during the five year period of observation, except for the slight postoperative rise for one day following operation.

On July 22, 1947 the frontal bone and orbit were exposed by an incision along the area of the orbital ridge. The incision was extended vertically at the outer margin to the hair line and a short curved incision was made downward over the lateral orbital rim. The scalp was retracted and the orbital contents were retracted downward and medially. The bone appeared healthy except for three small pieces which were embedded in scar tissue. These were removed. Two epidermoids were located. One was attached to the dura in the same area over the frontal lobe where the small round one had been removed at the previous operation; it connected with the fistulous tract to the exterior and was completely removed from the dura by blunt dissection. The other epidermoid, about the size of a marble, was situated below the frontal lobe far back in the upper lateral orbital wall. After removal of these growths, a tantalum plate was used to correct the defect in the orbital ridge. Iodoform gauze saturated with penicillin was inserted and brought out through the opening where the fistula had been excised.

A small amount of drainage persisted periodically for several weeks, but after this there has been no indication that there will be any further difficulty (fig. 4).

DIAGNOSIS OF EPIDERMOID OF FRONTAL BONE, SINUS AND ORBIT

The essential reason that persons with the diploic type of epidermoid of the skull and adjacent structures seek medical advice is the deformity produced by this slow-growing tumor. As this neoplasm enlarges, erosion of the bone occurs and orbital proptosis is produced from pressure, the extent of which depends on the size of the mass. The globe is depressed downward; there is limitation of upward rotation, and diplopia may be present. Reduction in visual acuity may take place as a result of pressure on the globe. Occasional headache may be noted after excessive use of the eyes. Symptoms referable to the central nervous system rarely occur. The tumor is usually firm. Roentgenograms show a characteristic round or ovoid defect with smooth borders. The patient otherwise appears perfectly healthy, with no past history or evidence of chronic sinusitis. Such tumors make their appearance relatively early in life and may be present many years before attention is given to them. Examination of the tumor contents reveals a laminated, rather firm, whitish yellow, cheeselike, waxy, odorless material of acellular character. Cholesterol crystals may or may not be present.

Differential Diagnosis.—Several conditions must be considered in differentiating the epidermoid growth from other slow-growing enlargements involving the frontal and parietal regions of the skull and orbit.

Skin Lesions: Sebaceous cysts are well encapsulated, freely movable, somewhat fluctuant common growths of the scalp. There is no bone defect, and this definitely differentiates the epidermoid from the sebaceous cyst.

Lesions of the Paranasal Sinuses: 1. Mucoceles, which are painless growths, produce bone erosion and orbital proptosis, as described by Chamberlin and Parry,[29] Collet and Charachon,[30] Sédan,[31] Wilkerson,[32] McNally and associates[33] and Alexander.[34] However, the fluctuant character of the mass, history of past sinusitis, mucoid content of the cyst and examination of the cyst wall will make the diagnosis clear.

2. Osteomas are easily differentiated by the dense bone formation, as shown by roentgenograms. Other characteristics have been thoroughly discussed by Gatewood and Settel,[35] Childrey[36] and Dowling.[37]

3. Malignant tumors grow more rapidly than epidermoids. Recurrent intranasal bleeding, hyperplasia of tissue and rapid regrowth after removal are diagnostic features. Early biopsy should substantiate the diagnosis.

4. Fibromas are exceedingly rare in this area, as described by Simpson and associates[38] and Woodruff.[39] A differential diagnosis may not be possible until operation.

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35. Gatewood, W. L., and Settel, N.: Osteoma of Frontal Sinus; Review of Literature and Report of Case Presenting Extended Invasion of Orbit, Arch. Otolaryng. **22**:154 (Aug.) 1935.

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37. Dowling, J. R.: Osteoma of the Frontal Sinus: Report of Five Cases, Arch. Otolaryng. **41**:99 (Feb.) 1945.

38. Simpson, W. L.; Graham, D. C., and Saunders, S. H.: Fibroma of Ethmoid and Frontal Region with Case Report, Ann. Otol., Rhin. & Laryng. **53**:344, 1944.

39. Woodruff, G. H.: Ossifying Fibroma of the Ethmoid Cells and Frontal Sinus, Ann. Otol., Rhin. & Laryng. **54**:582, 1945.

Lesions of the Orbit and Adjacent Tissue: 1. Dermoids, which contain deeper skin derivatives such as hair, are usually not so firm as epidermoids. Differential diagnosis may be impossible until operation, unless roentgenographic studies reveal radiopaque ectodermal derivatives.

2. Vascular tumors do not produce firm masses. There is very little to be felt in the orbit. Varicoceles produce intermittent exophthalmos when the head is lowered and then raised. Arteriovenous aneurysms produce a pulsating exophthalmos; a bruit is usually present. Compression of the carotid artery reduces the pulsation, bruit and proptosis.

3. Mixed tumor of the lacrimal gland may be differentiated by its position and mobility. If it is large and has invaded the surrounding structures operation and examination of the tissue may be required to substantiate the diagnosis.

4. Pseudotumors of the orbit develop more rapidly than epidermoids. No accurate outline of the tumor can be felt on palpation. There are no abnormal roentgenographic findings. Edema of the conjunctiva and lids may be present. Exploratory operation and biopsy show a chronic inflammatory reaction in the tissues.

5. Neurogenic tumors produce a gradual unilateral loss of vision, visual field disturbance and proptosis. Enlargement of the optic foramen can be demonstrated by roentgenogram, especially in neurofibroma of the optic nerve. In neurofibromatosis (von Recklinghausen's disease) there may be defects in adjacent bones and evidence of such tumors elsewhere in the body.

6. Fibroma of the orbit is exceedingly rare, very firm and cannot be diagnosed until operation, when its characteristic fibrous appearance will differentiate it from the epidermoid.

7. Lymphosarcoma usually produces a bilateral exophthalmos and is not an encapsulated lesion. Biopsy will reveal the characteristic cellular structure.

Intracranial Lesions Producing Exophthalmos: These are usually located in the middle or anterior cranial fossae. Symptoms referable to the central nervous system are usually present; they may be headache, disturbances of visual acuity and visual fields and paralysis of the third nerve. Findings on examination of the fundus may vary from bilateral stasis and hemorrhages to optic nerve atrophy. Roentgenographic studies show enlargement of the optic foramen and pituitary body and destruction of the pterygoid bone, depending on the location of the tumor. The commonest lesions are tumors of the pituitary gland and meningiomas.

Systemic Disorders and Metastatic Growths: 1. Thyrotoxicosis usually produces a bilateral exophthalmos. Although an increased basal metabolic rate, weight loss, tachycardia and heat intolerance are usually

present, the exophthalmos may precede the other signs of toxic goiter. No bone destruction can be demonstrated by roentgenographic examination.

2. Xanthomatosis may produce bone defects and orbital proptosis. In the typical Hand-Schüller-Christian syndrome, the skin, aponeurosis, visceral organs and bones are involved. Roentgenograms show the somewhat irregular bone defects. Diabetes insipidus is a prominent symptom. Biopsy will show the characteristic foam cells.

3. Lymphatic leukemia may be differentiated by the generalized lymphadenopathy, high leukocyte count, and a differential count revealing a pronounced lymphocytosis. Retinal and conjunctival hemorrhages occur. There is a pale milky appearance of the fundi and blood vessels.

4. Metastatic neurogenic growths from the adrenal and sympathetic ganglions produce proptosis and soft vascular tumors in the skull. Tumor masses are found in the regions of the kidney and liver. Roentgenograms reveal a worm-eaten appearance of the skull, long bones and ribs. Loss of weight, moderate anemia and intermittent fever are present.

5. Metastatic malignant growths of the prostate and the thyroid gland and of the lung and breast occasionally produce lesions in the skull and in the orbit. History and examination for tumors in these regions and biopsy will substantiate the diagnosis.

6. Tertiary syphilis may manifest itself in gumma formation involving the orbital apex or superior wall of the orbit. Impairment of one or more of the motor nerves of the eye is common. Proptosis of the globe and ptosis of the lid may be present. The usual symptoms and physical findings of luetic infection are to be found.

COMMENT

Several interesting features have been demonstrated by the case of epidermoid tumor presented in this paper.

The absence of any findings referable to the central nervous system is understandable, even though the tumor was lying against the dura. The bone destruction had allowed the growth to expand outwardly, the expansion producing orbital proptosis and external deformity of the skull. The outward expansion prevented intracranial pressure from developing. Consequently, there was no evidence of the type of headache, and other findings referable to the fundus and the central nervous system common to large tumors within the skull.

No diplopia occurred, even though pronounced downward displacement of the globe was present with the proptosis, because his vision was too poor for him to see distinctly with that eye. The reduced visual acuity was not due to amblyopia exanopsia, vascular or optic nerve involve-

ment, since the fundus was normal. I believe the gradual prolonged pressure from the tumor over a period of years produced enough change of contour of the globe to result in a greater error of refraction than that of the right eye. This is quite possible, since his visual acuity improved to 20/40 after removal of the tumor and was correctible to 20/20 with a + 2.25 sphere.

At the last operation an epidermoid tumor was found where one had previously been removed. This finding must mean that some of the wall of the first epidermoid growth was not completely removed. This recurrence emphasizes the importance of getting out all the tumor wall in order to prevent recurrence. Unfortunately, this is not always possible when the wall becomes too firmly attached to the dura mater. The other epidermoid tissue, which was found far back laterally and below the frontal lobe during the last surgical procedure, was an extension of the original massive tumor. Because of its inaccessible position, it was not discovered during the previous operation.

The larger such epidermoid growths become, the greater is the destruction of bone tissue and the greater is the distortion of the normal anatomy. This makes the removal of all the epidermoid tissue proportionally more difficult and hazardous.

One must not overlook the possibility of osteomyelitis developing after surgical procedures on the frontal sinus and adjacent bone. The periosteum should be conserved as much as possible. There was never any roentgenologic evidence of osteomyelitis during the five year period that this patient was under observation; nor did any clinical symptoms occur indicative of bone infection. However, some increase in the external deformity of the orbital ridge and the recurrent fistula formation cannot be disregarded as evidence of a low grade osteomyelitis, even though no sequestrum was extruded from the opening.

Some investigators reported a foul odor to the contents of tumors designated as epidermoids or primary cholesteatomas. The epidermoid growth presented in this paper was odorless. The so-called secondary cholesteatomas contain saprophytic and other organisms and may produce an odor. Otologists are aware of this finding. There is no logical reason that primary cholesteatomas or epidermoids should have any odor, and if an odor is present the growth cannot accurately be classified into this group.

This patient received an injury to the left brow at the age of 5 years, which may or may not have a bearing on the development of the tumor. Three possibilities must be considered: (1) The growth of the embryonic inclusion of epidermal tissue was precipitated by the injury; (2) the inclusion of epidermal tissue may have occurred at the time of injury, the continued growth later producing the epidermoid, and (3) the injury

was only incidental and bore no relation to the subsequent tumor. Several reports mention injury preceding the development of epidermoids of the skull. The possibility, therefore, cannot be ignored that injury in some way may play a part in the production of epidermoids of the skull.

SUMMARY AND CONCLUSIONS

Epidermoid tumors are rare neoplasms arising from misplaced or aberrant epithelial tissue. They are usually congenital but occur often enough after trauma for one to suspect that injury may play a part in the formation of such tumors.

The pathology, diagnosis, differential diagnosis and review of the literature on epidermoid tumors and so-called primary cholesteatomas of the skull, sinuses and orbit are presented.

A case report is included, in which the growth involved the frontal bone, sinus and left orbit. The so-called primary cholesteatomas are actually epidermoid tumors with a high cholesterol content.

Extradural epidermoid neoplasms involving the frontal bone, sinus and orbit are rather firm, slow-growing tumors that produce no symptoms. The cosmetic deformity is the chief reason that persons with this growth seek medical advice. A characteristic round or ovoid defect with smooth borders may be seen in roentgenograms of the skull.

The larger such growths become, the greater is the bone destruction and the more difficult they may become to remove entirely, especially if they are firmly adherent to the dura mater. The importance of removing all the tumor wall in order to prevent recurrence is stressed.

A low grade osteomyelitis must be watched for after operations on this region.

SARCOIDOSIS (BOECK'S SARCOID) OF THE UPPER RESPIRATORY TRACT

Report of a Case with Ten Years' Clinical Observation

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AND
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THE FIRST contribution to the medical literature on sarcoidosis of the upper respiratory tract was made by Poe.[1] The present paper is a continuation of the same case, covering a span of more than ten years. So far as we have been able to discover, this is the first record of clinical observations on sarcoidosis of the upper respiratory tract. The larynx will be described in considerable detail, since this organ in particular was watched during the entire period.

Since Boeck made his studies public, an exceedingly large amount of literature has accumulated on the subject. It is well known that the disease appears in many clinical forms. These forms have been well described at various times, and therefore we shall not discuss that aspect for the present. Since the historical development of the disease has also received much detailed treatment, it will not be given especial consideration here.

During the past few years a revival of interest in this singular ailment has been witnessed. Much writing has been, and continues to be, done on the subject. Yet, despite the large number of contributions, few can be found in the rhinolaryngologic literature. This paper is being offered in the hope that it might bridge at least a small portion of the gap.

REPORT OF CASE

V. S. was a fairly well nourished and normally developed Negro woman aged 36 when she was seen by us for the first time more than ten years ago. She stated at that time that her facial features had been regular and without abnormal discolorations prior to the appearance of skin blemishes, which persisted to a greater or less degree to the present. This condition suggested that infections took place at an early age and that their virulence had never completely abated. At times, the blotches, or pimples, attained large dimensions, and at other times would fade almost entirely from sight. At the initial examination, the patient was

1. Poe, D. L.: Multiple Benign Sarcoid of Upper Respiratory Tract, Ann. Otol., Rhin. & Laryng. **51**:430-444 (June) 1942.

fully aware of her facial disfigurement, conscious of stiffness in the fingers, an alteration in her voice and an inability to breathe through the nose. She complained also of shortness of breath when walking up an incline.

A cursory inspection of the skin of the face, nose, arms, fingers, hands and legs showed the characteristic infiltrations of sarcoidosis. The nodules and plaques were deep blue or violet and had a sharply defined border. They were round or oval and varied in size: Some were no larger than a pinhead and some were 1 to 2 cm. in diameter. On the upper portion of the legs and arms were large round or oval patches, which were rubbery to the touch, slightly thicker and considerably lighter than the surrounding skin. These patches were evidently the sites of old active lesions, but now they were dormant or entirely healed.

Examination of the nose revealed numerous distortions in the external architecture. The nose was feline in shape. The alae and tip were encrusted with discrete elevations or growths the size of grape seeds. The inferior border was irregular and bumpy. The interior presented many aspects of interest. On the anterior edge of the lower turbinate body, an elevation of pathologic origin, about the size of a large pinhead, was seen. On the septum, at the articulation of the vomerine cartilage, there was an irregular surface 1 cm. in diameter that appeared as if studded with worn hobnails. The lesions bled easily when palpated with a probe. Examination of tissue taken from this region and sent to the laboratory disclosed the presence of sarcoidosis.

Examination of the larynx showed that the epiglottis contained several nodules on the lingual surface. The right arytenoid cartilage was slightly edematous with minute nodulations. The ventricular fold on the left side was somewhat thickened. The vocal bands on both sides appeared normal and were of good motility. Subglottically, attached to the distal surface of the true cord on the left side, an irregular nodular mass with intact surfaces protruded into the lumen of the trachea. It occupied about a fifth of the free space of the upper portion of the trachea. On the anterior and the posterior aspect of the trachea, immediately below the aforementioned subglottic growth, were two fleshy irregular nodular masses with glistening unbroken surfaces. The masses were of such size that if they had been on the same level rather than one below the other they would probably have filled the entire lumen of the trachea. A bronchoscopic examination of the tracheobronchial region disclosed two or three suggestive discrete elevations near the carina. These were carefully removed with a biting instrument and forwarded to the laboratory for examination. They were found to be composed of the same histologic elements as the specimens taken from other parts of the body.

It was observed that when a good-sized portion of a growth was removed from an area, that part remained free from further infiltration for a long period and when new infiltrations appeared the regrowth was slow. Considerable time elapsed before the mass regained its former size. In the larynx, where the neoplasm showed a tendency toward becoming dangerously large, its bulk was reduced with biting instruments, and the raw edges were touched with 50 per cent solution of trichloroacetic acid. In this fashion the patient remained free from distressing symptoms referable to the respiratory system for several years.

In September 1943, during a demonstration before the otolaryngologic section of the American College of Surgeons at the New York Post-Graduate Hospital, this patient suddenly became alarmingly dyspneic. She was hastily removed to the operating room, and a tracheotomy was performed. With a tube in the trachea, the interior of the larynx was examined by mirror. The subglottic masses, which previously had been removed sufficiently to provide adequate breathing space, were now so closely approximated that only a comparatively solid tumor could be visualized. No effort was made to destroy or remove any of the pathologic tissue.

After the patient wore the tracheal tube for nine months, during which time the larynx remained at relative rest, the enlarged neoplastic body underwent certain retrogressive changes and shrinkage of the excessive bulk became visibly apparent. Free space was again sufficiently reestablished to serve as an ample airway. After the patient satisfactorily demonstrated that she could breathe freely naturally, the cannula was removed.

After two years of relative comfort signs of breathing embarrassment appeared. Apparently the friction produced by respiration, coughing and speaking, after the withdrawal of the tube, was sufficient to rekindle the growth of the pathologic

Fig. 1.—Specimen removed from the larynx; low magnification.

tissue. Again the fleshy mass of new growth took on added volume, and once more it appeared that surgical intervention would be necessary. However, reduction of the patient's physical efforts and enforcement of a regimen of silence brought about a reduction in the quantity and size of the morbid material, so that she was again able to carry on the duties of normal life. This play of natural forces continued on and off until a prophylactic tracheotomy followed by thyrotomy was performed. For approximately three months prior to operation we witnessed an example of nature's marvelous ability to adjust her needs to the capacity of attainment. The subglottic tumors had invaded the lumen of the upper parts of the trachea to such an extent that only a lengthy slit could be discerned during examination. With

eye measurement, the narrow opening was estimated to be 1.5 mm. wide and 0.5 cm. long. How a person with such little visible space for respiratory needs could carry on an apparently normal life, doing home chores, coming to and going from the clinic in a large metropolis distantly situated from her home and traveling among vast crowds is difficult to understand. But she carried on until she was persuaded to undergo a prophylactic tracheotomy.

In the previous description of the larynx, little was said about involvement within the organ. After the original condition was described, numerous imperceptible infiltrations occurred. The infiltrations, at first difficult to see, were

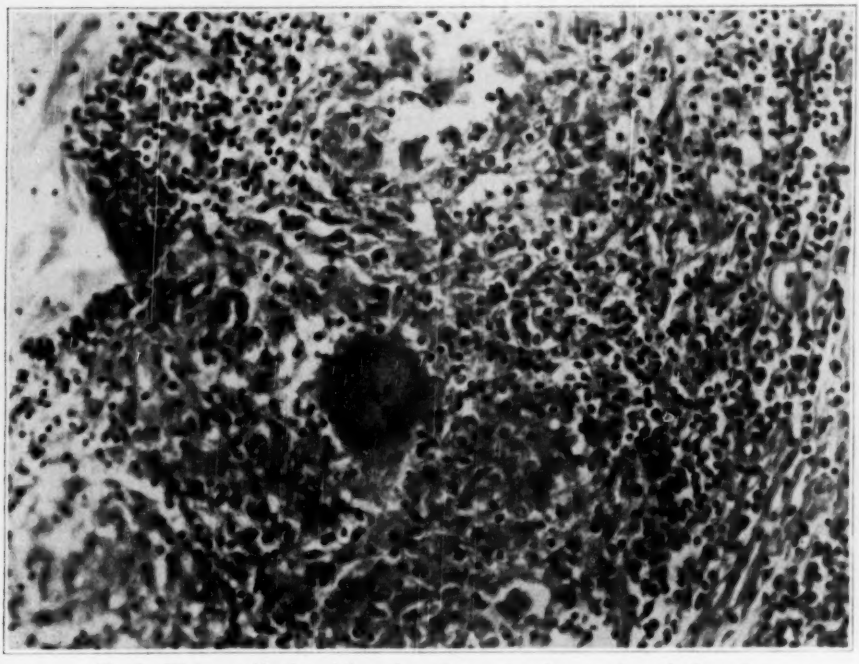

Fig. 2.—High magnification of tissue from the same specimen as shown in figure 1.

succeeded by others, thus coalescing to form a discrete nodule and then a patch with subsequent formation of a full tumor. At the close of 1945, almost the entire larynx was one mass of pathologic tissue. The ventricular bands on both sides were fleshy, brownish red hulks hanging over the true cords. The ventricles also were bulging forward, boggy and stiff. The vocal bands were almost entirely hidden from view. The small portions that could be glimpsed appeared whitish and possessed fair motility on phonation. The cricoarytenoid articulations were free. The movements were limited by being infiltrated with new tissue and the increased bulk of the membranes filling the interarytenoid space. Nowhere could any abrasions in the covering mucosa be detected.

At a clinical conference a laryngofissure was considered advisable, one side to be cleaned of the redundant material, with as much of the subglottic tumor removed as practicable. A thyrotomy was performed. At operation all the tissue was found to be exceedingly friable, tearing easily and bleeding copiously. Removal of the growth had to be done piecemeal. While it would have suited the operating surgeon to dislodge the whole mass and bring it out as one piece, it could not be done for the reason just mentioned. The vocal cords, after being freed, were found to be injected, similarly to the injection seen in a low grade chronic laryngitis. Of all the soft tissue entering into the formation of the larynx and upper portion of the trachea, the vocal bands undoubtedly presented the greatest resistance to the invasion of the disease. It was of interest to note that the pathologic invasions were confined almost entirely to the mucous membrane, the perichondrium having escaped involvement practically in its entire extent. All the material removed from the field of operation was immediately sent to the pathologic laboratory for examination. The findings were typical of sarcoidosis throughout.

The patient, having been under observation for a number of years, was often subjected to tests to determine whether at some period they might be positive for tubercle bacilli. Human, bovine and avian bacilli were used in different strengths, but the results were consistently negative. These tests were made from time to time because in the literature are reports of some positive reactions in some patients. Reports that tubercle bacilli were present in secretions of patients can also be found. While our bacteriologist made several earnest efforts to culture secretions for tubercle bacilli, the results were all negative.

A very interesting roentgenologic finding with reference to the mandible is worthy of mention at this time. A roentgenogram of the lower jaw disclosed a circular area of considerable size in the center of the horizontal portion of the bone. This finding was submitted as an original observation, no others having come to light, though zealous inquiry was made at sources considered most highly informed on matters of this nature. A detailed report of this observation will be found in the *American Journal of Orthodontics and Oral Surgery*.[2]

COMMENT

It is unfortunate that a diagnosis of this morbidity in its very early manifestation remains unrecorded. A better understanding of its early stages would be desirable. The condition in the majority of cases is in the stage of late development, and the patients present a tragic aspect. Because of insufficient knowledge of the various conditions making this phenomena possible, there is as yet no completely successful therapy. It is hoped that rhinolaryngologists will give more thought to the problem of sarcoidosis affecting the rhinolaryngologic region. Discoveries of new metabolic, biochemical or endocrine mechanisms may pave the way in the future for a therapy that will result in greater success.

Despite the lack of adequate information on the disease, much knowledge has accumulated. It is possible now in some cases to identify the morbidity long before the facial blemishes appear.

We were able to discover the presence of sarcoid in 2 patients who had no outward sign of changes in the skin and in 1 patient whose blem-

2. Poe, D. L.: Sarcoidosis of the Jaw, Am. J. Orthodontics **29**:52-56 (Jan.) 1943.

ish on the face, because of the history and the appearance, gave the impression of being a congenital nevus.

The first 2 patients complained of intermittent nosebleeds, and the third came to the Skin and Cancer Unit of University Hospital to have a facial nevus removed. This patient too had had occasional nosebleed.

A careful examination of the interior of the nose disclosed nodular infiltrations located on the septum in all 3 patients. The nodules were of pinhead size. The mucous membrane bearing the nodules was removed; in 2 patients a small portion of cartilage was also removed and sent to the pathologic laboratory for histologic examination. The cartilages were free from any invasion. The nodules showed the typical microscopic cellular structure which has come to be accepted as characteristic of sarcoidosis. This evidence alone, however, is insufficient to establish the diagnosis. The condition may be tuberculosis. It is imperative that one be differentiated from the other.

The first step toward establishing the diagnosis is to subject the patient to the old tuberculin test. The patient with sarcoidosis reacts negatively, while one with tuberculosis reacts positively.

The second step is to examine the chest roentgenologically. A roentgenogram of the chest of the patient with sarcoidosis frequently, though not always, will show a mass in the mediastinum, while in that of the patient with tuberculosis shadows of a mass are absent.

The fact that a relatively large mass exists in the chest indicates quite conclusively that the disease already is of a chronic nature and has been present quite some time, despite the absence of discernible skin lesions.

The third step is to undertake the Kveim test.

SUMMARY

A case of sarcoidosis of the upper respiratory tract is presented, with observation made over a ten year period.

The larynx in particular was involved, and a large obstructing mass necessitated a tracheotomy. Subsequently, the growth was removed from one side through a laryngofissure. In the trachea at the carina a nodule was removed which possessed the histologic characteristics of sarcoidosis. In the interior of the nose, nodular infiltrations of the nature of sarcoidosis were present.

Typical skin lesions were present on the face and hands.

Roentgenologically, a typical mass was found in the mediastinum.

There were also decalcified areas in some bones of the fingers, toes and mandible. A roentgenogram of the lower jaw revealed a particularly large rarefied area. This information is of unusual interest inasmuch as only one report of this case appears in the literature.

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Case Reports

ACUTE OTITIS MEDIA DUE TO KLEBSIELLA PNEUMONIAE TREATED WITH CHLORAMPHENICOL (CHLOROMYCETIN®)

ELMER A. FRIEDMAN, M.D.
CHICAGO

In the early days of use of any new therapeutic agent it is important that clinical experiences be reported and shared. The various reports add up to a wealth of knowledge, which makes easier the choice of treatment in similar cases. For this reason it is believed that the presented case is of interest.

Chloramphenicol (chloromycetin®) is one of the newest of the antibiotics and is already known to have one of the widest ranges of usefulness. Even for so new a drug a voluminous literature attests to its efficacy in the treatment of diseases caused by gram-negative bacteria, rickettsias and certain viruses. Among diseases caused by gram-negative bacteria, limited clinical study reveals that chloramphenicol is effective in the treatment of pneumonia caused by Klebsiella pneumoniae (Friedländer's bacillus). As far as can be ascertained, this is the first reported instance of its use in middle ear suppuration due to this organism.

REPORT OF CASE

The patient, an unmarried white woman aged 52, was admitted to the Michael Reese Hospital, on May 17, 1949, for emergency treatment in the care of a physician. A week preceding her admission to the hospital, otalgia developed in the left ear, followed by spontaneous rupture of the drum membrane and discharge from the ear. During that week she had chills, fever and malaise.

The patient was known to have a hypertensive condition and had diabetes of five years' standing. She had refused to take insulin, and the diabetes was controlled fairly well by diet. Twenty-two years ago she had had hemiplegia on the right and speech aphasia, which lasted for six months. Her speech had returned almost to normal, and there was only slight limitation of use of the right arm and leg.

Examination in the hospital revealed a very obese woman. The temperature was normal. The blood pressure was 160 systolic and 85 diastolic. The only other pertinent finding was that of purulent discharge from the left ear.

The results of the laboratory examinations were as follows: The Kahn reaction of the blood was negative; the blood sugar was 242 mg. and the blood urea was 16.6 mg., per hundred cubic centimeters. The red and white cell counts and the hemoglobin were within normal limits.

Roentgenograms showed moderate pneumatization of the mastoid processes, with decided clouding of the cellular system on the left side.

The patient was placed on a diabetic diet, and insulin and 300,000 units of penicillin in oil were administered daily. On May 19, two days after her admission to the hospital, against the advice of her physician, she signed her own release and left the hospital.

I saw her for the first time on May 25. There was a profuse pulsating discharge from the left ear and a prolapse of the membrana tympani. I felt that the patient's condition might very well be an otitis caused by Bacillus mucosus

capsulatus (Pneumococcus type 3) in a diabetic person. A culture of the discharge from the ear was taken, and she was given 300,000 units daily of penicillin in oil.

There was no improvement with this therapy. The discharge continued profusely, and there was increasing pain in the ear, especially at night. The aural infection was now of three weeks' duration, and clinical mastoiditis was definitely present.

On May 29, the bacteriologic report indicated that the culture yielded a pure growth of K. pneumoniae type A. A culture taken again on this day gave the same findings. Sensitivity tests gave the following results: streptomycin, 2.5 units; aureomycin, about 4.0 micrograms, and chloramphenicol, about 0.5 to 1.0 microgram.

On May 31 treatment was started with 750 mg. of chloramphenicol administered every six hours during the first day and 500 mg. every six hours thereafter. There was an immediate objective and symptomatic improvement. The discharge became less profuse, and the patient felt better. At the same time, she consented to take a daily injection of insulin. On June 6 the ear was dry and continued to remain dry, though the normal landmarks were not clearly visible until June 23. The chloramphenicol therapy was stopped on June 11, when there were no complaints other than stuffiness of the ear. The ear was inflated several times, and on June 30 hearing for the whispered voice was 12/12 in each ear. An audiogram, made on August 4, showed equal and approximately normal hearing in both ears.

COMMENT

Although it is true that good medical procedure dictates that before using an antibiotic or chemotherapeutic agent one should know the causative organism, it is also true that in the usual clinical practice this measure is rarely followed. Certainly, the majority of pediatricians, internists and otolaryngologists engaged in the treatment of complications of infections of the respiratory tract empirically use penicillin and sulfonamide derivatives almost exclusively. This practice is not wrong, for it is known clinically, as well as bacteriologically, that most of the organisms responsible for the common diseases and complications are sensitive to these agents. This case confirms, however, the fact that when a disease does not respond to specific therapy according to expectations, culture and sensitivity tests should be done immediately. Use of these measures is especially important now with the advent of so many new antibiotics, with their selective activity.

Most diseases are self limited, and one cannot say with certainty that in any particular case a happy outcome is due to the agent used and not to natural defensive forces. In this case, however, everything points to a specific antibiotic result. The progress of the disease during the first three weeks had been inexorable, and in the crucial fourth week the question of surgical intervention became paramount. Diabetic patients are recognized as having the severest complications of middle ear infections. In this case recovery was immediate and progressive after the onset of use of chloramphenicol.

55 East Washington Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

Review of 1947 Literature

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A LARGE part of the 1947 literature again is concerned with the surgical treatment of clinical otosclerosis. All significant contributions on the subject of deafness have been reviewed and arranged according to the following subjects: otosclerosis; deafness due to trauma; Ménière's syndrome, and miscellaneous contributions on causes and treatment.

OTOSCLEROSIS

Fowler [1] reports a detailed study of 5 pairs of identical twins with otosclerosis. In every instance menstruation began earlier in the twin now the deafer of the pair. In 3 pairs the phosphatase activity was lower in the deafer twin, and in 4 pairs it was lower in the twin who first noted any deafness. During several years little, if any, increase in deafness has been noted, in spite of pregnancies and infections of the upper respiratory tract in several cases. The deafness was shared by both twins of each pair with one exception. However, the deaf twin in this instance has only a 10 to 20 decibel loss in the worse ear.

Brickley [2] presents two case reports of van der Hoeve's syndrome—a triad of blue scleras, fragile bones and otosclerosis. The hereditary factor is traced through the male side in 1 case and the female side in the other. Deafness was initially manifested at the age of 19 and 29, respectively.

Moorhead [3] reports his experience with 123 cases of fenestration surgery. The series is consecutive and includes some cases with mod-

1. Fowler, E. P.: Otosclerosis in Identical Twins, Ann. Otol., Rhin. & Laryng. **56**:368-378 (June) 1947.

2. Brickley, D. W.: Otosclerosis and Blue Scleras, Arch. Otolaryng. **46**:230-236 (Aug.) 1947.

3. Moorhead, R. L.: Fenestration for Otosclerosis: Report of One Hundred and Twenty-Three Cases, Arch. Otolaryng. **45**:49-60 (Jan.) 1947.

erate and pronounced nerve degeneration. The period of nerve degeneration after operation varied from seven months to four years. The results were classified into three groups. In the first group were 57 patients (47 per cent), in whom the final threshold for all three speech frequencies was at or better than 30 decibels; in the second group were 19 patients (15 per cent), in whom the level reached 30 decibels in two of the three speech frequencies, and in the third group were 11 patients (9 per cent), in whom there was improvement above the 30 decibel level in only one of these frequencies. Hearing diminished below the preoperative level in 5 per cent. The use of a cartilaginous stopple in 24 patients did not significantly change the results. In answer to those otologists who state that a well fitted hearing aid will give as serviceable hearing as a fenestration operation, Moorhead questioned many patients who discarded their aids after operation and did not find one who would have preferred to remain as before operation.

[Reviewer's Comment: It is interesting to note that Moorhead stated that 3 of his most grateful patients were in the third group. This could be interpreted as further evidence that, from the patient's standpoint, the value of the operation cannot be judged entirely by merely determining whether the final level falls above or below an arbitrarily selected line across the audiometric chart. Factors such as the degree of deafness before operation, ability to use a hearing aid, the prospect of being able to continue in employment if an aid is used and other considerations enter into the patient's opinion about the success of the operation.]

Shambaugh [4] discusses the problem of evaluating the results obtained by fenestration surgery. Of 516 patients who, in answer to a questionnaire, regarded the operation as successful, 303 had reached the 30 decibel practical level or better. For estimate of the benefit to the patient, the decibel gain is important, as well as the decibel level attained. The decibel gain method is particularly useful for comparing technics. Of 415 patients operated on with use of the Northwestern University technic, which has been described previously, 390 (94 per cent) obtained an initial improvement of 11 decibels or more. This improvement was maintained at the two year postoperative period in 363 patients (or 93 per cent of the group with improvement). The incidence of bony closure has been reduced to less than 5 per cent by use of this technic. In a group of ideal patients (class A—good cochlear function) operated on in 1946, an initial practical (30 decibel or better) level was obtained in 83.6 per cent. A two year period of follow-up observation will be needed for final evaluation. In slightly more than 1,300 fenestrations done at the time of this presentation there have been no deaths or serious infections.

4. Shambaugh, G. E., Jr.: The Fenestration Operation: An Evaluation of Its Present Status, Surg., Gynec. & Obst. **84**:828-838 (April) 1947.

House [5] stresses particularly the use of the 1024 fork Rinne test in evaluating a patient with clinical otosclerosis for suitability for fenestration. He classifies a patient as ideal for the procedure if the threshold by bone conduction audiometry is not below 10 decibels for any speech frequency and if the 1024 magnesium alloy tuning fork is heard at least ten seconds longer by bone conduction as compared to the time it is heard by air conduction. A patient on the borderline has not more than a 20 decibel loss by bone conduction for two frequencies and not more than a 30 decibel loss for the 2048 frequency. The 1024 fork in these cases is heard five to ten seconds longer by bone conduction than by air. A patient is not suitable for operation if the results of the Rinne test are positive and if the loss by bone conduction audiometry is poorer than that of the patient on the borderline. Of 111 patients followed six months or more, 66 per cent have maintained practical serviceable hearing (79 per cent of the ideal and 55 per cent of the borderline cases). Closure of the fenestra has occurred in about 10 per cent.

Lindsay [6] reports his observations on results with 34 patients with consecutive fenestrations who were followed for more than eight months. Tests were made with pure tone audiometry and with a series of sixty monosyllabic words divided into low, medium and high pitched groups. Four patients had complete and 2 had partial closures. Of the patients in this series 76.5 per cent maintained a 30 decibel or better level of hearing. Failure in a few instances was obviously due to faulty selection of cases. Failure to obtain good hearing at the 2048 frequency was found to be reflected in the speech tests. Several patients maintained an excellent gain for the higher frequencies but had partial loss of the initial gain for the low tones. The speech gain in these patients was usually preserved, indicating that the 2048 and 4096 levels are more important for speech than those below the 1024. This loss for the low tones may be related to alterations in the physical characteristics at the fenestra. In this series, a persistent preoperative loss for bone conduction proved to be a reliable indication of decreased cochlear function and was consistently reflected in the surgical result.

Lempert [7] reviews his contributions to the surgical treatment of otosclerosis from the time of his original description of a one stage endaural technic up to the present. With his fenestra nov-ovalis technic he has been able to obtain initial improvement of hearing to a serviceable

5. House, H. P.: Indications for the Fenestration Operation, Arch. Otolaryng. **45**:312-318 (March) 1947.

6. Lindsay, J. R.: The Fenestration Operation: Observations on the Evaluation of Hearing Tests, Laryngoscope **57**:367-380 (June) 1947.

7. Lempert, J.: Lempert Fenestra Nov-Ovalis Operation for the Restoration of Serviceable Unaided Hearing in Patients with Clinical Otosclerosis: Its Present Evolutionary Status, Arch. Otolaryng. **46**:478-511 (Oct.) 1947.

level in about 80 per cent of the most carefully chosen suitable cases of clinical otosclerosis. However, in 25 per cent of these initially successful cases this improvement was maintained for less than two years, i. e., in a total of 60 per cent of the cases with operation, serviceable hearing was maintained for two years or more. The predominant problem in his experience is still that of maintaining patency of the fenestra. With reference to bone dust, he states that "neither the secondary removal of bone dust, nor the primary initial absence of bone dust, which needs no removal, can prevent osteogenesis from taking place in the freshly cut bony walls forming the rim of the fenestra."

On the basis of his observations of revisions and histologic studies on experimental fenestrations with monkeys, he believes that, given time, bony regeneration ultimately involves all three layers of the labyrinthine capsule; the repair of the endosteal layer is, as a rule, the first and fastest, whereas that of the endochondral layer is usually the slowest. His animal experimentations have not substantiated the belief that disturbance or removal of the endosteum facing the perilymphatic space beyond the fenestral region would necessarily result in the formation of new bone or that avoidance of injury to the endosteum would necessarily prevent bone regeneration in the perilymphatic space. He believes that adhesion of the membranous labyrinth to the tympanomeatal flap is not a means of preventing osteogenetic closure of the fenestra.

The former use and subsequent discard of the metal obturator and cartilaginous stopple are discussed. On the basis of two experiments with monkeys, to be described in a review of another article, he now hopes to prevent osteogenetic closure by burnishing the bony rim of the fenestra with pure lead.

In 1945 Cawthorne [8] was dissatisfied with his own results of fenestration and performed removal of the stapes in 20 cases. The functional results were variable but, on the whole, disappointing. After this procedure he returned to that of fenestration and improved his technic by using a Leitz dissecting microscope and taking more time to do the operation in order that each detail could be carried out with painstaking care. In cases of satisfactory suitability, about 50 per cent of the patients will permanently maintain a substantial improvement in hearing.

Passe [9] discusses his experience with fenestration surgery and summarizes it by stating that in 34 of 55 patients followed from six to twelve months, useful improvement in hearing had been fully maintained, but that complete assessment is not possible in less than two years. In

8. Cawthorne, T.: Review of Surgery of Otosclerosis, Proc. Roy. Soc. Med. **40**:320-328 (April) 1947.

9. Passe, E. R. G.: Fenestration Operation for Otosclerosis, Lancet **1**:171-174 (Feb.) 1947.

another paper [10] on otosclerosis, he reports that complete cessation of the pulsating type of tinnitus in a case was accomplished by cutting the preganglionic fibers of the first and the second thoracic nerve. He states that the sympathetic fibers which follow the vertebral artery may be particularly important.

Aubry [11] uses the endaural fenestration approach and performs an atticotomy without entering the antrum. With constant irrigation he enchondralizes and thins the margins of the fenestra. The area is contoured so as to facilitate close contact between the membranous labyrinth and the skin flap. In another article [12] he reports the findings of audiometric tests done during various stages of 18 fenestration operations. The first gain was noted when a fine opening into the semicircular canal was made. As the opening was enlarged, no further gain was noted. For the 1024 frequency and below, the gain averaged 16.3 decibels; above the 1024 frequency, the gain was 6.3 decibels. Only a very slight decrease in hearing occurred after the flap was placed over the fistula. A further gain of 5 to 10 decibels was observed a few months after operation.

Lempert and associates [13] report a large series of experiments with rhesus monkeys directed toward the problem of osteogenetic closure of the fenestra. In each monkey the experimental variation was done on one ear, and the other ear, for which the regular technic was used, served as a control. In the first group of 13 monkeys the endosteum was left intact in the experimental ear. Some were killed in a few days to study early changes in the labyrinth, and others were not killed until three weeks after operation. Free blood was noted in the fenestra early; then connective tissue proliferation, and, finally, osteogenesis. The experimental ear and control ear showed parallel changes. After twelve days all fenestrae showed unquestionable osteogenesis. In the second group, various metals were used to burnish the fenestral rim. The only fenestrae which remained patent were two that had been burnished with pure lead. The control ear in these 2 cases of patent fenestrae closed by osteogenesis. Tympanomeatal flaps were not used. Several stopple inserts were either partially surrounded or invaded by bone in such a manner as to suggest

10. Passe, E. R. G.: The Fenestration Operation for Otosclerosis, Post-Grad. M. J. **23**:507-516 (Nov.) 1947.

11. Aubry, M.: Surgery of Deafness, Ann. d'oto-laryng. **64**:5-25 (Jan.-Feb.) 1947.

12. Aubry, M.: Preoperative Audiometry: Evaluation on the Operating Table of Primary Gain in the Course of Fenestration, Ann. d'oto-laryng. **64**:167-168 (March-April) 1947.

13. Lempert, J.; Meltzer, P. E.; Schall, L. A., and Wolff, D.: Osteogenesis Following Fenestration of the Vestibular Labyrinth of the Rhesus Monkey: A Controlled Experimental Study, Arch. Otolaryng. **46**:512-527 (Oct.) 1947.

that in some mobility may have been interfered with. Osteogenesis was present in all fenestrae into or over which a fibrin film had been placed. As a result of experiments on 41 monkeys, the authors observed that the only fenestrae in which osteogenetic closure did not occur were the 2 fenestrae of which the rim had been burnished with lead.

Lindsay [14] presents further observations on factors influencing osteogenesis at the site of the labyrinthine fistula and, in particular, on the role of the covering flap. Several types of covering flap had been used with varying degrees of success in preventing bony closure in monkeys: a conjunctival graft and a full thickness graft of thin skin and periosteum obtained from the external meatus and the tympanomeatal flap, as used in the Lempert nov-ovalis technic. The tympanomeatal flap was most effective, although some patent fistulas were obtained by using the other tissues to cover the fenestra. Lindsay stresses again the need for contouring the semicircular canal so that the fenestra is on a convex surface, in order that the flap will establish wide contact with the membranous canal. He states that this is a very essential factor in maintaining patency and that if the flap pulls away from the fenestral surface, osteogenetic activity is more likely to ensue. Adequate initial packing is essential. Absence of infection after operation was probably also favorable toward maintaining patency. It was not possible to demonstrate any beneficial effect from roentgen radiation applied after operation. The "stopple" technic was applied in 14 ears. There was a pronounced tendency toward osteogenetic fixation of the stopple and a tendency toward osteogenesis and fibrosis in the perilymphatic space at the site of the fistula that was more than usual. Two patients are reported on in whom labyrinthotomy was done for unilateral Ménière's syndrome. The fenestra was left open and the postauricular wound closed. Both fenestrae have remained patent. Regenerated mucoperiosteum was found covering the fenestra in 1 patient at reoperation.

Maxwell [15] states, "The prerequisite training of the otosclerosis surgeon should include first of all a wide background of diagnostic and surgical experience in the general field of otology. . . . The training of the surgeon and the selection of the patient are two inseparable and eminently important problems in the over-all aspect of fenestration surgery." In testing, he stresses the use of tuning forks, particularly the 1024 Rinne and the 1024 and 2048 Schwabach. He asserts that it is absurd to conclude that a patient is severely handicapped or that the fenestration has not been successful if there is slightly more than a 30 decibel loss for just one or even two of the tones in the critical frequen-

14. Lindsay, J. R.: Healing of Experimental Labyrinthine Fistulas: Further Observations, Arch. Otolaryng. **46**:584-600 (Nov.) 1947.

15. Maxwell, J. H.: Training of Surgeons and Selection of Patient for the Fenestration Operation, Arch. Otolaryng. **46**:539-543 (Oct.) 1947.

cies, as recorded on the audiogram. If a patient who is handicapped by impairment of hearing has been rehabilitated by fenestration so that he can carry on normal group conversation the operation has obviously produced a good result.

Day,[16] in preparing the external meatus for operation, prefers not to use mercury or iodine solutions, because of an occasional sensitivity. He believes that the packing should not be inserted so tightly as to cause pain. Early ambulation after operation is highly desirable. A patient should not be operated on in the presence of uncontrolled allergy.

Meltzer[17] states that, in general, only those patients should be considered for revision who have obtained a serviceable level of hearing initially after fenestration and then, after several months, have gradually returned to their preoperative level. It is impossible to predict whether the closure is bony or fibrous. The best time for revision of the fenestra is usually six to twelve months after it has become evident clinically that the fenestra has closed; but this rule is not absolute. In the event that a stopple was used, earlier operation may be advisable and a revision may be considered, even though no initial improvement was obtained. The presence of a positive fistula response is not a reliable guide as to the functional patency of the fenestra. In well selected cases, restoration of hearing to a serviceable level may be anticipated in 20 to 25 per cent after revision. The danger of the hearing being made worse by revision is much greater than it was at the time of the original operation.

Hershey and Jones[18] discuss the desirability of providing anesthesia for fenestration in a manner that will minimize bleeding, eliminate unpleasantness to the patient and, at the same time, avert the hazards inherent in heavy preoperative medication. Their present routine is described: The premedication consists of a light dose of pentobarbital sodium followed by morphine sulfate and scopolamine hydrobromide. Basal narcosis is then induced with tribromoethanol U. S. P., and the patient is taken to the operating room as soon as he becomes drowsy in order that oxygen may be administered well before the full effect of the basal narcosis occurs. An adequate airway is maintained by inserting both an oropharyngeal and nasopharyngeal tube after cocainizing the pharynx lightly. Inhalation anesthesia with nitrous oxide is used throughout the operation. Electrical equipment may be used safely in the presence of nitrous oxide.

16. Day, K. M.: Medical and Surgical Care of the Patient Selected for Fenestration of the Labyrinth, Arch. Otolaryng. **46**:534-538 (Oct.) 1947.

17. Meltzer, P. E.: Revision of the Fenestration Operation, Arch. Otolaryng. **46**:528-533 (Oct.) 1947.

18. Hershey, S. G., and Jones, M. F.: Anesthesia for the Surgical Correction of Deafness, Arch. Otolaryng. **46**:390-394 (Sept.) 1947.

Whitaker and associates [19] describe an endaural incision which differs chiefly from the original one described by Lempert in that the third incision is omitted, thereby preserving the original posterior meatal wall. By tucking this back into position on completion of the endaural operation, the circumferential integrity of the posterior edge of the meatus is maintained, and the normal epithelial covering decreases the tendency toward granulations and stenosis at this point. A composite diagram of the entire sensory innervation of the external meatus is presented. Adequate local anesthetic block, particularly of the meatal branches of the auriculotemporal and the meatal branch of the vagus nerve, together with general infiltration, has eliminated the need for the heavy sedation formerly used. Consequently, postoperative depression is now no longer a surgical hazard.

Brunner and Cutler [20] report their observations concerning nystagmus and labyrinthine symptoms in 12 cases of fenestration. Twenty-four hours after fenestration there is horizontal and rotatory nystagmus of the third degree to the side of the operation; it disappears, as a rule, in three or four days. In contrast to this, after surgical injury of the horizontal semicircular canal in the presence of infection, the direction of the nystagmus is to the opposite side. Slow oscillatory movements of the eye, synchronous with the pulse, are sometimes observed and are not considered indicative of infection. If, however, the nystagmus is directed after fenestration toward the side not operated on, or if it persists for more than a week, it may be indicative of infection. The authors state that a more accurate determination of the functional patency of the fenestra can be made by testing the fistula response by compression and by aspiration of the air in the external auditory canal (with a Politzer bag) than by the usual method of probing with a cotton applicator.

Ersner and Saltzman [21] propose a simple method of speech testing to be used to supplement other tests in evaluating cases of otosclerosis to determine suitability for fenestration. Two lists of words are used; one contains words of the lower frequency range, and the other contains words with frequencies above 2500 cycles. The patient is seated 4 feet (122 cm.) from the examiner, lip reading not being permitted. The relative percentage of words of low and high frequency repeated correctly is then determined. The examiner uses average conversational intensity if the patient has a 35 or 40 decibel loss and a louder voice if

19. Whitaker, C. F.; Juers, A. L., and Shambaugh, G. E., Jr.: A Modified Endaural Incision with Nerve Block Anesthesia for Fenestration and Mastoidectomy, Arch. Otolaryng. **45**:662-667 (June) 1947.

20. Brunner, H., and Cutler, M. H.: Labyrinthine Symptoms Subsequent to Fenestration of the Labyrinth, Arch. Otolaryng. **45**:613-618 (June) 1947.

21. Ersner, M., and Saltzman, M.: Speech Hearing in Otosclerosis, Arch. Otolaryng. **46**:753-761 (Dec.) 1947.

the deafness is more than 45 or 50 decibels. Patients with good cochlear function get a relatively higher percentage score for the high frequency words than they do for the low frequency words; and those with nerve involvement have a higher score for the low frequency words than for words of the high frequency. The audiometer microphone offers a further means of determining which patient is suitable for fenestration. If microphone amplification greatly improves the hearing for speech the chances are that a fenestra nov-ovalis procedure will do likewise.

DEAFNESS DUE TO ACOUSTIC TRAUMA

Graebner [22] presents a survey of the audiometric findings of a group of 449 airline pilots of various ages and air hours' experience. All the pilots had flown the same type of plane. Their ages ranged from 21 to 53 years. The graph lines did not show any consistent tendency toward notching, but, rather, a steady decline toward the higher tones. This trend became more pronounced with increase in both age and air hours flown. The loss for the high tones was definitely more than that noted in comparable age groups not exposed to noise. There was no apparent hearing loss for conversation; hence, the need for careful, periodic audiometric examination for ascertaining that high tone signals will be heard. Tinnitus in these cases was seldom observed.

MacLaren and Chaney [23] discuss an analysis of acoustic trauma in 5,000 cases during the past four years in an air frame manufacturing establishment. A study of acoustic spectrums showed that pure tones or narrow frequency bands were not usually present, the noise, in general, covering practically the entire audible range. The maximum over-all loudness from various sources were as follows: drop hammers, 130 decibels; heavy riveting, 126 decibels, and speed hammers, 110 decibels. The initial loss was usually at the 2896, 4096 and 5792 frequency levels. The usual individual variation in susceptibility to trauma was observed. The dividing line between innocuous and hazardous noise was found to be somewhere between 70 and 100 decibels. Susceptible persons manifested most of their loss during the first three or four weeks, and subsequent change was very gradual. One group of workers with a moderate early loss showed considerable recovery when they wore protective devices in the same noise environment. The authors state that the best attack on the problem should be directed toward control at the noise source. The possibilities here are somewhat limited. Protective devices are effective but frequently are discarded by workers.

22. Graebner, H.: Auditory Deterioration in Air-Line Pilots, J. Aviation Med. **18**:39-47 (Feb.) 1947.

23. MacLaren, W. R., and Chaney, A. L.: An Evaluation of Some Factors in the Development of Occupational Deafness, Indust. Med. **16**:109-115 (March) 1947.

Preemployment audiometric studies and rechecks at intervals may serve as a basis for detecting persons susceptible to trauma.

Taylor [24] describes a newly designed ear defender called the V-51R ear warden. It affords acoustic insulation of from 25 to 30 decibels at the low end and 40 decibels or more at the high end of the audiofrequency spectrum.

Hoople, Wolfe and Bregande [25] report their observations on audiometric studies performed on 1,200 soldiers who had been in active combat but had had no complaint of loss of hearing. A composite audiogram made of this group showed that for the frequencies from 2896 to 11584 there was an approximate loss of 10 to 15 decibels more than shown on a similar study of a comparable group of 150 soldiers who had not been exposed to battle noise. Below the 2896 level, essentially the same curve was shown for the two groups. The authors stress the need for routine audiometric testing at the time of induction if accurate evaluation of service-connected disability is to be made later.

Grove [26] reviewed 832 cases of craniocerebral trauma with demonstrable signs of injury and found that in 27.5 per cent impairment of hearing developed. In some younger persons tinnitus was the initial symptom and the hearing was not impaired until weeks or months later. The hearing loss was predominantly of the perceptive type. Older people seemed to be more vulnerable. Deafness may be unilateral or bilateral. A severe, subtotal loss of hearing may occur without any demonstrable evidence of bone fracture and is probably due to hemorrhage into the inner ear. Follow-up examinations on some patients indicated that a traumatically produced hearing defect has just as much chance of remaining stationary or improving as it has of further deterioration.

MÉNIÈRE'S SYNDROME

Cawthorne [27] discusses in considerable detail the symptomatology and pathogenesis of Ménière's disease. He points out that "in established cases the diagnosis was rarely in doubt. The paroxysms of vertigo alternating with intervals of freedom, the perceptive deafness, almost invariably more noticeable in one ear, and the absence of any signs of disorder within the central nervous system presented a characteristic picture that was seen in all the cases in this series." For conservative treatment he has found that the exclusion of salt and salty

24. Taylor, M.: Ear Defenders, Laryngoscope **57**:137-141 (Feb.) 1947.

25. Hoople, G. D.; Wolfe, W. C., and Bregande, S.: Unrecognized Battle Noise Trauma, Laryngoscope **57**:125-136 (Feb.) 1947.

26. Grove, W. E.: Hearing Impairment Due to Craniocerebral Trauma, Ann. Otol., Rhin. & Laryng. **56**:264-270 (June) 1947.

27. Cawthorne, T.: Ménière's Disease, Ann. Otol., Rhin. & Laryng. **56**:18-38 (March) 1947.

foods from the diet, together with a limitation of the fluid intake, is most satisfactory. In some cases histamine injections have been used. While a focus of infection has always been searched for, it has not been found to be very important in his cases. Patients whose attacks were not controlled by conservative treatment, in whom only one vestibular labyrinth was affected and in whom the hearing on the same side was obviously impaired were considered suitable for destruction of the end organ by operation. Of 135 patients so treated, 120 improved and were able to work; 9 did not improve and were unable to work, and sufficient follow-up observations were not available for 6 after operation. He suggests a program of rehabilitation, which includes a series of special head exercises to facilitate readjustment to the absence of one labyrinth.

While this symposium [28] does not lend itself to abstracting without losing its value, it deserves being mentioned as an excellent source of information for any one interested in the physiology, symptomatology, pathology and general neurologic aspects of vertigo.

Williams [29] gives a comprehensive review of all aspects of endolymphatic hydrops from the time of Ménière's original paper up to the present. He discusses in particular the probability that the histopathologic picture of hydrops is the end result of an allergic phenomenon, which, in most instances, is of the physical or intrinsic type. Most of the effective treatments proposed have been directed toward correcting the electrolyte imbalance in the tissue fluids, toward controlling fluid metabolism or toward stimulating the autonomic nervous system and producing vigorous vasodilatation. While it is believed that sensitization in physical allergy occurs more readily in the presence of infection, experience at the Mayo Clinic has not indicated that focal infection is very often an etiologic factor in such patients. The most satisfactory treatment in the experience of physicians cited by Williams has been the use of histamine or nicotinic acid. As to surgical treatment when indicated, labyrinthotomy seems preferable to section of the eighth nerve. In the historical review of the bibliography, Williams points out that Lake in 1910 reported complete relief from vertigo in 10 cases of endolymphatic hydrops, in which the lateral semicircular canal and the vestibule had been opened through a transmeatal approach on the affected side and in which, in addition, the stapes had been removed.

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28. McNally, W. J.; Lewy, A.; Lindsay, J. R.; Levy, I.; O'Leary, J. L., and Furstenberg, A. C., in a symposium on Vertigo, Ann. Otol., Rhin. & Laryng. **56**:514-582 (Sept.) 1947.

29. Williams, H. L.: The Present Status of the Diagnosis and Treatment of Endolymphatic Hydrops (Ménière's Disease), Ann. Otol., Rhin. & Laryng. **56**:614-646 (Sept.) 1947.

Lewy and Fox [30] report the use of pyridoxine for treatment of vertigo due to Ménière's syndrome or of undetermined cause. It was used empirically with a fair degree of success. No statistical analysis of results is given.

Gooch and Hallberg [31] report a case of Ménière's disease in a deaf-mute. The usual medical measures were ineffective. There was very little response to caloric tests of both static labyrinths. Since the patient was completely incapacitated for work, a bilateral labyrinthotomy was done. The patient made good compensation, and nine months later reported that he was again able to work.

MISCELLANEOUS CONTRIBUTIONS ON CAUSES AND TREATMENT

Dunlap and Schuknecht [32] describe closure of perforations of the tympanic membrane with repeated applications of trichloroacetic acid to the margins of the perforations. The effectiveness of treatment is dependent on cauterizing often enough to maintain a raw granulating border, thereby enabling the collagen fibers to contract gradually and to form a bridge for the epithelium to advance on. A few drops of saturated solution of urea are instilled twice a day when activity seems to be slow. In most cases at least several months are necessary to accomplish closure.

Goodhill [33] calls attention to the urgent need of starting the special education of the severly deafened or completely deaf child as soon as the handicap is recognized. If the child has mastered a fair degree of speech reading before entering a special school at 6 years, a tremendous step has been accomplished toward enabling that child to establish itself and to participate eventually in a speaking society. Parents of deaf children need to be educated concerning their responsibilities and potentialities in starting this training during the most important formative preschool years. The John Tracy Clinic is mentioned as having pioneered in this field.

Pattee [34] reviews the embryology of the ear as related to atresia of the external auditory meatus. He has conceived a new endaural surgical approach, in which a meatus is created and a substantial improvement in

30. Lewy, A., and Fox, N.: Pyridoxine (B6) Used in the Treatment of Vertigo, Arch. Otolaryng. **46**:681-683 (Nov.) 1947.

31. Gooch, J. O., and Hallberg, O. E.: Surgical Treatment of Ménière's Disease in a Deaf-Mute, Proc. Staff Meet., Mayo Clin. **22**:252-254 (June) 1947.

32. Dunlap, A. M., and Schuknecht, H. F.: Closure of Perforations of the Tympanic Membrane, Laryngoscope **57**:479-491 (July) 1947.

33. Goodhill, V.: The Educational Treatment of the Pre-School Deaf Child, Laryngoscope **57**:555-562 (Aug.) 1947.

34. Pattee, G. L.: An Operation to Improve Hearing in Cases of Congenital Atresia of the External Auditory Meatus, Arch. Otolaryng. **45**:568-580 (May) 1947.

hearing is attained. Atresia is usually associated with microtia. The condition may be bilateral or unilateral. If it is unilateral the hearing result in the ear operated on cannot approach that of the normal ear, but operation is advisable from the cosmetic and psychologic standpoint. Operation should not be done until it has been determined that sufficient cochlear function is present. At operation an endaural incision is made and the soft tissues freed over the mastoid process; this, in itself, frequently permits the microtic appendage to fall back into a more normal position. There is usually no landmark to identify the position that the external meatus should have. The cortex is entered well posteriorally, and the antrum and lateral semicircular canal are identified. The malleus and incus are usually fused. They are both removed, leaving the stapes freely mobile. The cavity is then lined with a split thickness skin graft. A report on 5 patients is included, showing good hearing improvement in 4 and some improvement in the fifth.

A surgical technic is described by Ombredanne [35] for fenestration of the lateral semicircular canal for improvement of hearing in cases of congenital atresia of the external auditory meatus. The fenestra is covered with a skin graft. Two cases are reported in which a pronounced hearing improvement was obtained. [Reviewer's Comment: If the stapes is freely movable, merely establishing a patent external auditory meatus by the method of Pattee should suffice. If the stapes is fixed, an additional fenestration of the horizontal semicircular canal by the method of Ombredanne is indicated.]

Goodfriend [36] presents a rather lengthy discussion on the development of symptoms such as deafness, tinnitus, vertigo and neuralgia as a result of malocclusion and on their correction by restoring normal temporomandibular function through dental treatment. He reports 3 cases in which treatment was successful. He states, "These cases and my clinical experience during the past twenty-one years show that proper dental treatment will cure certain patients with aural vertigo and neuralgia. It will also alter the intensity and the type of tinnitus in certain patients and make it more acceptable to them. In certain patients it will alleviate deafness of short duration and retard the progress of hearing loss in others." [Reviewer's Comment: While temporomandibular dysfunction may be more important in the genesis of otologic disturbances than is generally appreciated, it is to be regretted that the author did not provide more concrete and audiometric evidence, substantiated by otologic diagnosis, to justify his seemingly optimistic

35. Ombredanne, M.: Surgery of Deafness: Fenestration in Cases of Congenital Atresia of the External Auditory Canal, Oto-rhino-laryng. internat. **31**:229-236 (Nov.) 1947.

36. Goodfriend, D. J.: Deafness, Tinnitus, Vertigo and Neuralgia, Arch. Otolaryng. **46**:1-35 (July) 1947.

viewpoint about the efficacy of his treatment. His statement that dental treatment will "retard the progress of hearing loss in some cases" must be viewed with some skepticism, inasmuch as no one can predict that deafness in a given case would have progressed if no treatment had been applied.]

Kelly and Langheinz [37] stress again the necessity of considering the dynamics of the temporomandibular articulation in evaluating dysfunction, because approach to the problem only from a static concept might end in failure. Of 18 cases of neuralgia, there was complete relief in 14. In 7 cases the symptoms were (*a*) stopping or stuffy sensation of the ears, most noticeable about mealtime; (*b*) tinnitus, usually buzzing, and (*c*) occasionally a snapping noise with chewing. The condition was unilateral in 6 cases, with relief in all. In 2 cases of glossodynia no relief was obtained. No improvement in hearing audiometrically was observed in any instance.

Pohlman [38] describes his experience with a prosthesis inserted into the external meatus to restore useful hearing in patients in whom the conduction mechanism was absent, owing to previous otitis media. The suitability of a patient is first found by testing the stapes and round window area with an "acoustic probe" to determine if a sound receptive spot is present. The prosthesis must be individually fitted and is based on the pattern of the middle ear structure in the bird. Several representative audiograms are shown, indicating, in a few instances, improvement of as much as 30 to 40 decibels. Hearing improvement to a useful level was obtained in a few cases in which the fenestration had not improved the hearing.

Tamari and Speas [39] report their otologic findings with 398 children in a school for the deaf. It was determined that 289 were deaf or had defective hearing on a congenital basis. Vestibular response was routinely checked with turning tests. Caloric tests were done if there was no response to turning. The vestibular response was positive in 87 per cent of the children with congenital deafness, and no reaction was elicited in 13 per cent. Of the 63 children whose deafness or defective hearing was considered as being due to other causes (i. e., chronic otitis media, measles, etc.), 67 per cent presented normal vestibular responses and 33 per cent showed no response. In the meningitic group of 46, a

37. Kelley, W. J., and Langheinz, H. W.: Dental Treatment of Trismus, Tinnitus Otalgia and Obscure Neuralgias, Arch. Otolaryng. **45**:191-204 (Feb.) 1947.

38. Pohlman, M. E.: The Artificial Middle Ear, Ann. Otol., Rhin & Laryng. **56**:647-657 (Sept.) 1947.

39. Tamari, M., and Speas, R.: Ear, Nose and Throat Survey of Children in the School for the Deaf at Jacksonville, Illinois, Ann. Otol., Rhin., & Laryng. **56**:425-436 (June) 1947.

normal reaction was noted in 32.8 per cent and no reaction in 67.2 per cent. Tinnitus was present only in the postmeningitic group. Good usable speech was present in 20.6 per cent of the total group. These children had either large hearing residuals or were in the postmeningitic group. Of the group, 9.8 per cent had only fair speech. The rest had very poor or no speech at all.

Clayton-Jones [40] made a survey in England in some schools for the deaf to determine whether rubella in the mother during early pregnancy was a probable cause of deafness in some of these children. At one school with 18 children, 8 mothers gave a definite history of rubella in the second to fourth month of pregnancy. In three other schools the incidence of rubella in the mother during early pregnancy was 11 out of 123. Nearly all these children were born between August and October of 1940. Records revealed an epidemic of rubella in this area early in 1940, in which many young women had the disease. The deafness in the children was bilateral, incomplete and showed a rather uniform loss in all frequencies. No cataracts or definite cardiac lesions were detected.

Fuchs and Almour [41] report a case of allergic deafness. The patient experienced repeated attacks of rather sudden deafness with intervals of normal hearing. On three occasions a slow subcutaneous injection of epinephrine relieved the condition within half an hour. Injections of isotonic sodium chloride solution were ineffective. An allergic cause was demonstrated. There were no vestibular symptoms. Recognition of this type of deafness depends on the following factors: (1) a history and clinical proof of allergy; (2) the absence of organic pathologic conditions in the ears; (3) periodic occurrence of deafness with normal intervals; (4) ability to reproduce the allergy by exposure to the allergen, and (5) immediate relief by injection of epinephrine.

For evidence of aural toxicity, Fowler and Seligman [42] studied 81 patients treated with streptomycin. The usual dosage was 3 Gm. daily. In most cases the total dosage varied from 10 to 49 Gm. Caloric stimulation after streptomycin therapy revealed a normal response in 65 (80.2 per cent); a "delay" in one or both sides in 13 (16 per cent), and an absence of response in 3 (3.7 per cent). The authors comment that a high incidence of vestibular disturbance and a sizable number of cases of deafness, either transitory or permanent, will occur with the use of streptomycin if large doses of the drug are given over prolonged periods. As a rule, the onset of otic symptoms is on the seventeenth to

40. Clayton-Jones, E.: Rubella as a Cause of Congenital Deafness in England, Lancet **1**:56-61 (Jan.) 1947.

41. Fuchs, A. M., and Almour, R.: Allergic Deafness: Report of Case, New York State J. Med. **47**:1397-1398 (June 15) 1947.

42. Fowler, E. P., Jr., and Seligman, E.: Otic Complications of Streptomycin Therapy: Preliminary Report, J. A. M. A. **133**:87-91 (Jan. 11) 1947.

twentieth day if 3 Gm. per day are given. The patient seems to recover from the deafness even though the caloric response remains negative. Compensation for the loss of labyrinthine function occurs to a remarkable degree. Tinnitus may be an indication of impending deafness.

Horton, Hallberg and Williams [43] used histamine intravenously to treat patients with nerve deafness, particularly those with sudden onset without vestibular symptoms. In 9 such patients, the duration of deafness was twenty-four hours to six months; 4 obtained excellent restoration of hearing; 3 showed some improvement, and 2 showed no improvement. Best results were obtained in those with deafness of shortest duration. Another group of patients had sudden nerve deafness associated with acute infectious processes. Only 1 of these obtained an excellent result. Two patients with nerve deafness secondary to head injury showed no improvement. Of 7 patients with gradual onset of nerve deafness, 2 showed excellent results. Of 18 patients with Ménière's syndrome, 8 showed decided improvement. In general, the good results were obtained in those with deafness of shortest duration. [Reviewer's Comment: The mechanism of the restoration of hearing with histamine therapy is not known. Before histamine therapy can be accepted as useful for nerve deafness, the indications for it should be clearly defined.]

43. Horton, B. T.; Hallberg, O. E., and Williams, H. L.: Nerve Deafness: Treatment by Intravenous Histamine, Proc. Central Soc. Clin. Research **19**:79-80, 1946.

News and Comment

SCHOLARSHIP FOR STUDENTS OF LIP READING

The American Hearing Society announces that applications are being received for the Kenfield Memorial Scholarship for 1950, which amounts to $100.00 Applications for the scholarship will be considered from any resident of the United States who desires to teach lip reading (speech reading) to the hard-of-hearing with or without other types of hearing and speech therapy. Those persons already teaching lip reading cannot be considered.

The applicant should be a college graduate with a major in education, psychology, and/or speech, together with professional training in lip reading: 30 clock hours of private instruction under an approved teacher of lip reading or 60 clock hours of instruction in public school classes under an approved teacher of lip reading.

The winner of the scholarship may take the training course from any normal training teacher, school or university in the United States offering a course acceptable to the teachers' committee of the American Hearing Society. The scholarship must be used within one year from the date the award is made.

Applications must be filed between March 1 and May 1, 1950, with Miss Rose V. Feilbach, chairman of the teachers' committee, 1157 North Columbus Street, Arlington, Va.

CORRECTION

In the article by Dr. Kurt Tschiassny, "Herpes Zoster Oticus," which appeared in the January 1950 issue of the ARCHIVES, page 80, line 17, the word "phonophia" should read "phonophobia."

Abstracts from Current Literature

Ear

HOME TRAINING FOR PRESCHOOL DEAF CHILDREN THROUGH CORRESPONDENCE. HARRIET ANDREWS MONTAGUE, J. Speech & Hearing Disorders **14**:131 (June) 1949.

The home correspondence course from the John Tracy Clinic, of Los Angeles, for parents has been mailed over a period of six years to more than 2,000 families. Sixty-five per cent of those who enrolled completed the course. About 75 per cent finished at least the first six instalments with varying benefits. The author states that good results were obtained by those who utilized the course conscientiously and intelligently.

PALMER, Wichita, Kan.

A SPEECH AND HEARING PROGRAM FOR CHILDREN IN A RURAL AREA. HARRIET M. DUNN, J. Speech & Hearing Disorders **14**:166 (June) 1949.

This article is a review of a speech and hearing program in rural Vermont. Diagnostic speech and hearing clinics are held weekly in four cities accessible to rural areas. Follow-up work, including certain types of medical care, and provision of transportation to classes help in solving certain social problems. Classes in speech correction and auditory training are held three days a week at the center in Rutland, Vermont. An eight week intensive training program is carried on in a summer residence center for children with speech and/or hearing disorders.

PALMER, Wichita, Kan.

A SAFE AND EFFICIENT CLEANSING DEVICE FOR THE NASOPHARYNGEAL RADIUM APPLICATOR. SAMUEL FOX and G. BROOKS WEST JR., Laryngoscope **57**:339 (April) 1948.

The authors describe a device which is a cylindric, thick-walled brass container, 17 cm. in height and 4 cm. in diameter, with an opening extending down the center for the radium applicator. Bristles for cleaning the applicator protrude from its sides. The device is filled with a fresh solution of caroid,® a protein digestant, and benzalkonium chloride U. S. P. (zephiran chloride®), an antiseptic.

HITSCHLER, Philadelphia.

RADIOLOGIC STUDY OF THE MASTOID BONE IN INFANTS IN CONJUNCTION WITH ANATOMIC AND PATHOLOGIC INVESTIGATIONS. HENRY G. KOIRANSKY, IRVING K. ETTMAN and EDWARD L. WHITE, N. Y. State J. Med. **49**:1291 (June 1) 1949.

The authors undertook to determine roentgenologically the earliest age at which definite mastoid cells are present. They made roentgenograms of infants from birth to 1 year and determined that the mastoid process is absent or minute at birth. A divergence of opinion with Cheatle, Albrecht and Wittmaack, who are quoted, is manifest: These authors "noted that pneumatization of the mastoid began to appear at about six months" and was definite after seven months. However, there was inequality on the two sides in many patients. A study of a stillborn child weighing 15 pounds (6,804 Gm.) and delivered by cesarean section was made, first roentgenographically and then with stained sections in series

after decalcification; various "outpointings" from the mastoid antrum into the mastoid process were observed, which were regarded as primary mastoid cells with a lining membrane continuous from the middle ear cavity. From this the authors conclude that "cellular invasion may occur even in the newborn." Bacterial invasion can also take place by the same route; hence, the value of early administration of antibiotics in all acute infections in infancy and childhood, a factor well understood by pediatricians. The conclusion is that formation of mastoid cells is more a matter of physical development than of the chronologic age of the individual child. Six references to the literature and 1 figure are included.

VOORHEES, New York.

CLINICAL VESTIBULAR DISTURBANCES DUE TO STREPTOMYCIN. JEAN BOUTIER, Rev. de laryng. **70**:244 (May-June) 1949.

Observation of 73 patients treated with streptomycin for pulmonary tuberculosis during 1948 showed that vertigo and imbalance developed in 39 of them; 13 patients had no vertigo, but vestibular tests showed inexcitability or more or less pronounced hypoexcitability. Twenty-one patients showed no subjective or objective vestibular findings. The ages of the patients had no influence on the appearance of the vestibular disorder. Hypoexcitability of the labyrinth was determined by caloric and rotation tests; results of the galvanic tests were normal. The subjective symptoms (vertigo) lasted several days to several months; even after the cessation of vertigo most patients retained the imbalance when their eyes were closed and when they were in the dark. The authors stress the importance of this factor in patients' occupational life. The imbalance without visual control persisted for a long period, particularly in cases in which the labyrinth was completely inexcitable.

No hearing loss was observed in the 73 cases reported. As for treatment, only thiamine appeared to have a beneficial effect. Reduction of the dosage of streptomycin from 2 to 1 Gm. daily avoids vestibular effects.

HERSON, Chicago.

Pharynx

THE USE OF SODIUM PENTOTHAL ANESTHESIA IN ADULT TONSILLECTOMIES. IRL H. BLAISDELL, Laryngoscope **59**:721 (July) 1949.

Thiopental sodium U. S. P. (pentothal sodium®) was used in 183 consecutive cases of tonsillectomy on adults with success. Its advantages are obvious and many. Its disadvantages include more profuse bleeding during operation and the possibility of laryngospasm. The latter danger can be almost eliminated by the procedure outlined in the article. Thiopental sodium is recommended highly for tonsillectomies on adults.

HITSCHLER, Philadelphia.

VINCENT'S ANGINA—TREATMENT WITH PENICILLIN BY ATOMIZER. A. E. FISHMAN, Med. Rec. **161**:356 (June-July) 1948.

A simple and pleasant method of local administration of penicillin to buccal mucous membrane for the treatment of Vincent's angina is described. A solution of 100,000 units to 30 cc. of distilled water or isotonic sodium chloride solution is sprayed into the mouth and on the gums by ordinary atomizer every two hours. Twenty patients were treated, of which only 4 required treatment longer than four days for complete recovery. The described method is particularly advantageous for use in children because of the ease of administration as compared with topical application and the use of mouth washes, injections and lozenges.

FERDINAND, Peoria, Ill. [AM. J. DIS. CHILD].

THE PAEDIATRIC APPROACH TO TONSILLECTOMY. J. ALISON GLOVER, Arch. Dis. Childhood **23**:5 (March) 1948.

Despite criticism, the incidence of tonsillectomy, the most frequent cause of admission to the hospital, remains excessive, particularly in children between 5 and 7 years. The most reliable indication for operation is the occurrence of frequently repeated attacks of acute tonsilitis which cannot be explained by extraneous infection. The operation has no value as a prophylactic measure against common infectious diseases, with the possible exception of diphtheria. In residential schools with high tonsillectomy rates, while the incidence of recurrent sore throats may be somewhat diminished, that of frequent colds is slightly increased. The incidence of otitis and mastoid disease is the same, or perhaps slightly increased, in the tonsillectomized children, while their liability to bronchitis and pneumonia is decidedly increased. The operation is never urgent and the procedure should always be preceded by an observation period of six months after the completion of any necessary treatment of teeth or sinuses. The operation should not be done in the winter or early spring, during a prevalence of infectious diseases, such as measles or influenza, or especially when poliomyelitis is epidemic.

KELLY, Milwaukee, [AM. J. DIS. CHILD].

Larynx

IS LOCAL TREATMENT OF TUBERCULOUS TRACHEOBRONCHITIS JUSTIFIED? J. P. SECRETAN, Pract. oto-rhino-laryng. **10**:36, 1948.

The author raises an important question about the value of local intratracheal treatment of tuberculous tracheobronchitis. He outlines the various methods of treatment, mentioning actual cautery of the tuberculous ulcerations and local application of silver nitrate of various strengths. He stresses the danger of complications that may arise, such as hemorrhage and cicatricial stenosis. Secretan describes 2 cases in which treatment had been given and concludes that local treatment is of value but must be supplemented by early collapse therapy.

ABRAM H. PERSKY, Philadelphia.

PERFORATION OF THE ESOPHAGUS BY FOREIGN BODIES, AND EMERGENCY TREATMENT. J. P. TAILLENS, Pract. oto-rhino-laryng. **10**:215, 1948.

Taillens discusses the dangers of esophageal perforation by foreign bodies, the principle danger being that of a mediastinitis, which is almost invariably fatal. This complication is not due to trauma, per se, but to secondary infection carried in from the digestive tract. He advises an immediate emergency gastrostomy, following the extraction of the foreign body by the esophagoscope.

PERSKY, Philadelphia.

CAUSTIC STENOSIS OF THE ESOPHAGUS. AUGUSTO LATORE AGUERO, Rev. otorrinolaring. **7**:81 (Sept.) 1947.

Caustic esophagitis is a frequent occurrence in Chile and is a symptom of a disease that represents one of the most serious social problems, namely, alcoholism. Most of the patients treated were alcoholic addicts, and the substances used most frequently were hydrochloric acid and sodium hydroxide. The author has treated 41 patients within the past four years. His method was usually that of early dilation with mercury bougies, starting either on the fifth or sixth day after the onset

of the condition. Of the group of 41, 11 patients died and 14 had to have a gastrotomy performed; in 8 patients pulmonary tuberculosis developed. The author is of the opinion that prognosis depends on the immediate institution of emergency treatment, proper dilation and the maintenance of a diet sufficiently rich in proteins to maintain body weight.

PERSKY, Philadelphia.

DISTURBANCES OF PHONATION OF THE LARYNX. ALEJANDRO MARAMIO, Rev. otorrinolaring. **8**:126 (June) 1948.

The causes of disturbances of the function of phonation fall into three groups: (1) diseases of the phonation system itself, (2) disturbances of the auditory apparatus and (3) local and general factors which may secondarily affect the phonation system. The first and third groups of causes produce either organic or functional disturbances, while the second group of causes produce only functional disturbances.

Organic disturbances of phonation can be produced by myositis or atrophy of the muscles, due to inflammatory processes of the larynx, either acute or chronic. They may be due to paralysis of the various muscles. The cause of paralysis of the recurrent nerve may be malignant tumors of the mediastinum; cancer of the thyroid; aneurysm of the aorta; sequelae to pericarditis; mediastinal adenopathies; hypertrophy of the left auricle, associated with mitral stenosis; pleuropulmonary mediastinitis; diabetes; disorders of the thyroid, such as thyroiditis, abscess and intracystal hemorrhages; post-thyroidectomy and ligature of the superior thyroid artery. Paralysis may also be associated with various neurologic syndromes.

Treatment of voice alteration due to organic disease is essentially treatment of the causative factor. Phoniatrics tends to establish by means of exercises new muscular adaptations or to improve the normal muscular functions. True functional disturbances of the voice are those in which organic lesions are not factors; they involve mainly alteration in muscular tonus. If muscular tonus is exaggerated the impulse is increased; this increase produces a hypercontraction of the muscles involved in phonation. On the contrary, if the tonus is decreased there is hypocontraction. If there is inequality in the tonus of different muscles there is lack of coordination between the sounds produced. Phoniatrics is thus a study to evaluate either hypertonicity or hypotonicity of the various muscles involved in speech production and takes for its role the establishment of an equilibrium between expiration, laryngeal muscular activity and the adaptation of the resonant cavities to pitch and sound. Various methods of treatment must be employed, depending on the pathologic condition present. In certain cases (hypercontraction), treatment consists of vocal rest plus massage and electrical treatment with either galvanic or faradic current, heat, ultraviolet irradiation and use of antispasmodics and calcium. On the other hand, when there is hypocontraction or myasthenia, treatment consists of vocal exercises, muscular massage, faradic and galvanic current, and use of strychnine and vitamin B preparations. Treatment is always a long, slow, tedious process.

PERSKY, Philadelphia.

RESULTS OF TREATMENT WITH STREPTOMYCIN IN EIGHTY-FIVE PATIENTS WITH TUBERCULOUS PHARYNGOLARYNGITIS. GUY ARNAUD, Acta oto-rhin-laryng. belg. **2**:3, 1948.

In a series of 85 patients, 67 were treated with intramuscular injection of streptomycin and 18 with streptomycin in aerosol alone. All patients had open tuberculosis with positive sputum.

Results of Treatment with Streptomycin Administered Intramuscularly.—Sixty-two patients had relief from or disappearance of the dysphagia and could therefore feed themselves and gain weight. In 46 patients the hoarseness improved, with

recovery of the normal voice in 20 patients. There was regression of pathologic lesions in the throat and lungs to the extent that there was total recovery in 23 patients and partial recovery in 33; there was no change in 6, and the condition was aggravated in 11.

Among the clinical forms of tuberculosis of the laryngopharynx were miliary (10 patients, with recovery in 4, improvement in 5 and aggravation of the condition in 1); infiltroedematous (19 patients, with recovery in 9, improvement in 9 and aggravation of the condition in 1), and ulcerative and hypertrophic, vegetative (32 patients, with recovery in 10, improvement in 19 and aggravation of the condition in 3). Pulmonary associated lesions in the miliary form benefited from the streptomycin much more than lesions in the ulcerative and infiltrative forms.

All patients received 1 to 1.50 Gm. of streptomycin daily. No definite relation between the amount of streptomycin given and the degree and speed of recovery was observed. Some patients received 50 Gm., others 100 or 200.

Results of Treatment with Streptomycin in Aerosol Alone.—Of these patients, the dysphagia subsided in 6, improved in 6 and did not change in 5; the hoarseness improved in 7, and the pathologic lesions improved only in 1. At each session of treatment, made once daily, Arnaud used 0.25 to 0.50 Gm. of streptomycin.

In conclusion, he obtained much better results with the streptomycin administered intramuscularly than with it in the aerosol. The latter method is considered as accessory treatment. Intolerance to streptomycin occurred often with this method. Of the patients thus treated, 10 showed laryngeal irritation, cough, dyspnea and, sometimes vertigo. The reactions occurred generally after the eighth treatment.

HERSON, Chicago.

Nose

THE NASAL TIP IN ROUTINE RHINOPLASTY. L. W. EISENSTODT, Eye, Ear, Nose & Throat Monthly **26**:372 (July) 1947.

The author stresses the importance of correction of the nasal tip in all cases of rhinoplasty, and reviews the Aufricht method, which is in common use. He presents an excellent restatement of commonly utilized procedures, and photographs add to the general interest of the report. SELTZER, Philadelphia.

REFINEMENTS IN PLASTIC SURGERY OF THE NOSE AND EAR. R. A. FRANKLYN, Eye, Ear, Nose & Throat Monthly **26**:419 (Aug.) 1947.

Franklyn discusses briefly a method for the correction of prominent ears, which does not vary notably from other methods in routine use. He expresses a preference for a solid plastic substance, such as methyl methacrylate, for repairing a saddle nose and correcting a receding chin, over either preserved or autogenous cartilage. He has seen no foreign body reactions to these agents. Four photographs accompany the paper. SELTZER, Philadelphia.

PLASTIC SURGERY OF THE FACE WITH ESPECIAL REFERENCE TO THE NASAL HUMP. L. W. NABERS, J. Tennessee M. A. **41**:1 (Jan.) 1948.

This paper is a good general review of the subject of rhinoplasty. It includes a brief history of the subject and comments on common nasal deformities and on submucous resection. The author discusses the preoperative measures and the choice of an anesthetic, and warns of a poor outcome if only one deformity is corrected when more are present. In conclusion, he states that good surgical judgment and careful operative technic are necessary for successful results.

SELTZER, Philadelphia.

Miscellaneous

INFLUENZA VACCINE: A STUDY OF SEROLOGIC RESPONSES AND INCIDENCE OF REACTIONS FOLLOWING SUBCUTANEOUS AND INTRADERMAL INOCULATION. HENRY B. BRUYN, GORDON MEIKLEJOHN and HENRY BRAINERD, Am. J. Dis. Child. **77**:149 (Feb.) 1949.

The authors, who are from the Infectious Disease Laboratory of the San Francisco Hospital, investigated the relative merits of subcutaneous and intradermal methods of vaccination against influenza with a combined type A and type B vaccine prepared from chorioallantoic fluid of chicken embryo. Seventy-nine children were inoculated. Of the group inoculated subcutaneously, one-half the subjects had a temperature of 101 F., usually with malaise, headache and vomiting. In the group inoculated intradermally with 0.1 cc. in one dose or 0.2 cc. in two doses, the reactions were one half as frequent as in the group with subcutaneous inoculation. The conclusion is that "the intradermal route . . . in children, especially when two doses of 0.1 cc. each are given three days apart, produces increases in antibody comparable to those in adults and is accompanied with greatly lowered incidence and severity of reactions as compared with the subcutaneous route."

The intradermal route of inoculation was suggested by L. Tuft (*J. Lab. & Clin. Med.* **16**:552-556 [March] 1931); its merits have been confirmed in the use of scarlet fever, diphtheria and typhoid toxoids. Navy researchers reported good results with few unfavorable reactions from intradermal administration. Bruyn and his associates offer nine tables and twenty-six references in explanation of their researches. [ABSTRACTER'S COMMENT.—For over fifteen years, I have used "cold vaccines," either autogenous or stock (the former preferred), *intradermally*. A series of six or eight inoculations in the fall and spring plus, of course, some local treatments, has helped to keep patients free from colds.]

VOORHEES, New York.

HAND-SCHÜLLER-CHRISTIAN DISEASE and EOSINOPHILIC GRANULOMA OF THE SKULL. HAROLD F. SCHUKNECHT and HENRY B. PEARLMAN, Ann. Otol. Rhin., & Laryng. **57**:643-676 (Sept.) 1948.

Destructive lesions of the skull occurring as a primary feature in a disease may present a diagnostic problem, and Hand-Schüller-Christian disease with its related conditions must be considered. Letterer-Siwe disease and eosinophilic granuloma are intimately related to the former disease and are considered to be clinical gradations of expression of the same basic disorder underlying the Hand-Schüller-Christian disease. Letterer-Siwe disease is a severer type and eosinophilic granuloma a milder.

The former condition is rapidly fatal, usually occurring before the age of 2 years. The principal clinical findings are enlargement of the liver, spleen and lymph glands; secondary anemia, purpuric eruption, destructive skeletal lesions, especially in the skull, and a rapidly downhill, acute, febrile course.

Hand-Schüller-Christian disease usually appears in childhood but may occur in the second or third decade or later. The mortality rate is about 30 per cent. Initial symptoms may be exophthalmos, diabetes insipidus or tender areas over the skull. Stunting of growth, involvement of the ear and mastoid process, anemia, pathologic fractures, hypogenitalism or adiposity may be first to appear. Infiltration of the liver, spleen, heart, lungs, meninges, spinal cord and brain may occur. Cutaneous eruptions showing characteristic lipophages may also occur. Eosinophilic

granuloma (solitary granuloma of bone) is usually seen in children or young adults. The long bones, skull, ribs and vertebrae are most commonly involved. Local pain and tenderness are the usual symptoms, without accompanying visceral, neural or cutaneous involvement.

When small, the lesions of the temporal bone are silent, but as they grow they may perforate the wall of the external auditory canal, erode through the cortex of the mastoid, the zygomatic or squamous portions of the bone, invade the labyrinth, involve the facial nerve and encroach on the structures in the jugular foramen, and the temporal bone may become secondarily infected. Uncomplicated lesions of the Hand-Schüller-Christian disease and eosinophilic granuloma of the temporal bone are almost always painless. When granulations appear in the external auditory canal, they arrive by passing through the canal wall and not through a defect in the tympanic membrane. Diagnosis can usually be made by microscopic examination of the tissue. When secondary infection occurs the primary condition is often overlooked. When the nature of the disease is suspected it is important to seek roentgenographic evidence of other skeletal or visceral lesions. Mastoidectomies sometimes must be performed in the event of complicating infection, and operation should be followed by roentgen ray therapy, as the wounds heal slowly if this is not done. Positive diagnosis by biopsy must be made before radiation is given.

In eosinophilic granuloma there is usually a solitary lesion, though there may be several. The lateral supraorbital region of the frontal bone and adjacent roof of the orbit appear to be the site of predilection. Either surgical removal or irradiation of an eosinophilic granuloma results in clearing of that lesion.

Seven cases are reported, in 3 of which there had been initial destructive lesions of the temporal bone, and in 4, solitary granuloma of the skull. In making the diagnosis, the following conditions should be considered: metastatic osteolytic malignant tumor, primary carcinoma, secondary cholesteatoma, primary cholesteatoma, osteomyelitis, multiple myeloma, congenital dehiscences of the skull, tuberculosis, syphilis and fungous lesions. MILLER, Philadelphia.

BOECK'S SARCOID. CURRENT COMMENT, J. Am. M. A. **135**:228 (Sept. 27) 1947.

Boeck originally described sarcoid as a disease that is characterized by multiple nodular lesions of the skin of unknown cause, associated with superficial lymph node enlargements. This conception has been extended to include similar, if not identical, changes in practically every organ in the body. Histologically, the changes bear a noteworthy resemblance to those of tuberculosis. Giant cells in the tubercle-like formations of sarcoid contain deposits of calcific material, such as are seldom, if ever, encountered in the giant cells of tuberculosis. Giant cells are not always present in the histologic lesions of sarcoid, and when they are present, do not always contain calcific inclusions.

In 1941, Longcope reported that "from inoculation of tissue into guinea pigs, rabbits and pigeons, we have not obtained any evidence to support the view that the disease is caused by human, bovine or avian tubercle bacilli."

The view that Boeck's sarcoid is a form of tuberculosis seems to rest solely on microscopic differences in the giant cells, occasioned by the deposit in them of calcified material in sarcoid and its absence in the giant cells of tubercles.

GORDON, Philadelphia.

Bronchiectasis Following Aspiration of Timothy Grass. Max G. Carter and Kenneth J. Welch, New England J. Med. **238**:832 (June 10) 1948.

Eight cases of pulmonary disease are presented, each due to the aspiration by children of timothy grass heads. In all but 1 of these cases, lobectomy was required, but in the 7 instances recovery from a formidable procedure was complete. There was no characteristic bronchoscopic appearance, nor was the bacteriologic flora consistent. Bronchiectasis and abscess were the most prominent pathologic findings, while the major symptoms included cough, loss of weight, abdominal pain and fever. Of especial interest is the delay from the time of aspiration of the grass to the final diagnosis, a confirmation of the need for awareness.

Gengenbach, Denver [Am. J. Dis. Child].

Migraine Headache Treated Successfully by Head-Traction Manipulation and Thiamin Chloride. Murray M. Braaf, New York State J. Med. **49**:1812 (Aug. 1) 1949.

Braaf read this paper before the Society of Alumni of Lebanon Hospital, Feb. 8, 1949. In it he stated that irritation of the roots of cervical nerves causes pain, to be sure, but one must keep in mind the possibility of herniation of the nucleus pulposus of the cervical intervertebral disk with pressure on nerve roots. Communicating branches when stimulated may cause pain in the head, neck and ears. Trauma to the neck may be so slight as to be unnoticed or forgotten, and yet it may cause migraine. Also constant straining of the neck, as in reading or awkward bodily positions, may induce pain. Other factors such as allergy, alcohol, menstruation, emotional upsets, severe coughing and barometric changes may be culpable. Heredity is another possible basic cause. Treatment by traction plus administration of thiamine hydrochloride has given good results in 20 cases. Sayre's traction apparatus was used to stretch the neck muscles and cervical portion of the spine, applied with the patient recumbent on a hard table for five to ten minutes. From 300 to 400 Gm. of thiamine hydrochloride was given intramuscularly after manipulation. Procaine hydrochloride solution (2 per cent) may be injected into the painful area. Diagnosis is based on the occurrence of head pains (usually hemicrania), periodicity of attacks, gastrointestinal symptoms, cortical disturbances, scotoma, photophobia, paresthesias, etc., and relief through ergotamine tartrate (gynergen®) or its allies. Patients who had had these violent attacks for less than five years had better prospects for cure than did patients with chronic migraine.

Voorhees, New York.

Hay Fever as a Public Health Problem. Leopold C. Lazarowitz, New York State J. Med. **49**:1960 (Aug. 15) 1949.

Working in the division of allergy at the New York Post-Graduate Medical School and Hospital, Lazarowitz has concerned himself with the economic losses due to hay fever and with its prevention and alleviation. "The importance of hay fever as a public health problem has not yet been fully appreciated." Some 3,000,000 persons are attacked in Greater New York each year between about August 15 and October 1. Hay fever predisposes to other allergies, notably seasonal asthma and sinusitis, with loss of sleep, anorexia, asthenia and poor general health. Inheritance is an important factor (predisposition), and it is questionable whether a man and woman both of whom are allergic should marry! So-called hay asthma is an extremely undesirable complication. Treatment is, of course, preventive; namely destruction of ragweed which is now an intensive

effort of the New York City Board of Health. The antihistamines help, but their use is limited to palliation chiefly. A "chilling method" is being tried at the Post-Graduate Hospital. "Damage to health, loss of time and money, and predisposition to upper respiratory diseases and bronchial asthma should arouse the attention of socially minded physicians and public health authorities. . . . The economic gain . . . will outweigh lost time and . . . expense," if the public at large can be taught to cooperate, especially in prevention. VOORHEES, New York.

USES AND ABUSES OF PENICILLIN. PAUL A. BUNN, New York State J. Med. **49**:2059 (Sept. 1) 1949.

From the Syracuse University College of Medicine, Bunn states that "for therapy of infections caused by most gram-positive and a few gram-negative organisms, penicillin continues to be the most effective, the most powerful, and the safest antimicrobial agent that has been given to the physician." The one drawback is its relative ineffectiveness against many gram-negative germs. The aim in therapy is to concentrate a maximum dose of penicillin at the site of infection and to maintain it at a given level as long as necessary. Because of rapid elimination of the drug, it has been difficult to maintain a continuous optimal blood level, but in the relatively insoluble salt procaine penicillin combined with either the sodium or potassium salt of penicillin G the benefit of delayed removal is provided. Penicillin G in aqueous solution given in large doses intramuscularly twice or thrice daily works well in the therapy of many acute infections. Large doses at widely separated intervals are now routine in management of infections due to highly sensitive organisms.

Abuses are too common in penicillin therapy. Some 60 per cent of all hospital patients receive penicillin regardless of their need for it. "Penicillin is not a routine diagnostic tool and its indiscriminate administration for fever alone or before an accurate diagnosis is made, can only be condemned." All antimicrobial therapy is merely a front line of defense to hold out against the enemy until the natural body forces take over. "Penicillin should not be used as a substitute for thinking." One must not "mask" the diagnosis by too early or too excessive use, save in extreme instances when life is in danger. VOORHEES, New York.

INFECTIOUS CROUP: I. ETIOLOGY. EDWARD F. RABE, Pediatrics **2**:255 (Sept.) 1948.

This report is based on a study of 347 cases of infectious croup. Infectious croup is defined as a disease of the upper part of the respiratory tract characterized by inflammation of the rhinopharynx with downward spread of the infection, leading to variable degrees of stenosis of the laryngeal, laryngotracheal or laryngotracheobronchial air passages. The stenosis is due to edema and is occasionally aggravated by formation of membrane.

On the basis of recognized signs and symptoms, infectious croup has been divided into three clinical groups, laryngitis, laryngotracheitis and laryngotracheopneumonitis. Such a classification proved to have prognostic but not etiologic significance for the individual case. By correlation of the bacteriologic and pathologic aspects and clinical course of the present cases, infectious croup was divided into three distinct etiologic categories: diphtheritic croup, Hemophilus influenzae type B croup and so-called virus or nonbacterial croup.

FROM THE AUTHOR'S SUMMARY [AM. J. DIS. CHILD].

FACIAL CLEFTS AND THEIR SURGICAL MANAGEMENT IN VIEW OF RECENT RESEARCH. W. B. SLAUGHTER and A. G. BRODIE, Plast. & Reconstruct. Surg. **4**:311, 1949.

On the basis of the findings of more recent research on the relation of facial clefts in the embryo to later growth of the face, Slaughter and Brodie present a theory, supported also by their own surgical experience, that the older method of early correction of the bony defect in cases of cleft palate and cleft lip is responsible for later deformity. By the means of roentgenograms and the use of dyes, a study of the postnasal growth of the maxilla has shown that this change is the determining factor in the final positioning of the parts of the maxilla and of the form of the mature face. There is general growth of the face until about 5 years of age. Thereafter, facial growth continues in the sutures and synchondroses of the skull, by proliferation of cartilage. This growth about the maxilla controls the final position of the parts of the face and is consequently concerned in the occurrence of cleft palate and cleft lip. The authors are of the opinion that injury from early operation on the bony margins of the maxilla, where later postnatal growth occurs, may destroy this potentiality, with consequent lack of normal relations of these parts. They have shown by their own experience that early corrections of only the soft parts averts disturbance of normal growth of the bone, leaving the action of the muscles and skin to mold the bony relations of the cleft palate and lip. The text is well illustrated by drawings and photographs, and sixteen references to related literature are given.

SELTZER, Philadelphia.

DERMAL GRAFTS FOR CORRECTION OF FACIAL DEFECTS. W. W. SCHUESSLER and D. N. STEFFANOFF, Plast. & Reconstruct. Surg. **4**:341, 1949.

In reporting their study of the correction of disturbances of the contour of the face in 80 cases, Schuessler and Steffanoff review the various materials which have been used for this type of reconstruction. The topology and histology of the skin are reviewed in the consideration of dermal grafts. The authors consider that dermal grafts have a definite place in plastic surgery of the nose and face. In this type of graft there is at first a volumetric increase from the vascular hyperplasia that occurs. This is followed by a later decrease in volume of the graft. In their experience, complication arose in only 7.5 per cent of their series, and of these, epidermoid cyst formation took place in only 1.25 per cent. The Zintel split-split method is given preference because of the ease and uniformity in securing the graft. Details are given of the two operative methods employed in this series of operations. Advantages and disadvantages in the use of dermal grafts are considered, and a tabulation of the complications arising in 6 cases is presented. The article is illustrated with one drawing and nine excellent photographs. An adequate bibliography is appended.

SELTZER, Philadelphia.

PRIMARY CLOSURE OF HARELIP. V. P. BLAIR and R. R. ROBINSON, Surg., Gynec. & Obst. **86**:502 (April) 1948.

In this article the authors deal editorially with the subject of cleft lip. They point out that freshening and uniting of the border of a cleft does not usually give satisfactory results. The reason for the operation is that it provides free breathing and assures that the lip will be symmetric. Deformity is caused partly by some reduction in the amount of lip tissue in all instances but chiefly by tissue displacement. The first step is to mark out a pattern for incisions that will allow adjustment of the soft tissues without much tension. There is no single rule for doing

this, since conditions are seldom exactly alike. An operation done early, during the first two days of an infant's life, is simple, and at two weeks thereafter the infant will present a nearly normal appearance, which is gratifying to the parents. Secondary operation may be needed some time during the first two years of life. If the primary operation is delayed until later childhood, there may then be a greater deformity that will require more extensive operation for correction, with a greater degree of undermining and removal of segments of both skin and cartilage to raise the displaced alae to the proper position. If the defect is not corrected earlier, at adolescence deflection of the nose may necessitate an extensive plastic operation. Many methods have been used for correcting cleft lip and palate, but the authors suggest means for closing complete double cleft lip and palate in two steps: 1. The premaxilla should be corrected by the Rose or Mirault method to give good occlusion of incisors and suitable contour of face. 2. The shortening of the nose resulting from the push-back of the premaxilla in closing the hard palate should be corrected by raising the superficial lip flaps and separating, at the same time, the short columella from the septum. Detailed directions are given for this operation, with the reminder that if the operation is not carefully performed from the beginning, the final result will be unsuccessful; but if it is properly performed, the outcome should be satisfactory.

SELTZER, Philadelphia.

OTOLOGIC EXAMINATIONS IN HEPATITIS EPIDEMICA. J. VARGA, Acta oto-laryng. **36**:356 (Dec.) 1948.

The author states that involvement of the eighth nerve, especially of its cochlear branch, in virus diseases is not an infrequent complication. Epidemic hepatitis, however, is not usually accompanied with neurologic lesions. In Varga's series of 30 patients, cochlear lesion was observed in 3 patients and vestibular damage in 1. The histories of 2 patients indicated disease of the middle ear; the history of the third patient, involvement of the inner ear and migraine attended with vestibular phenomena.

It is the author's opinion that the site of reduced resistance does, in addition to the virus infection, play a decisive part in localization of these complications.

ROBERT LEWY, Chicago.

THE QUESTION OF PARAPLEGIA AFTER LUMBAR APPLICATION OF PENICILLIN. ROBERT HLADKÝ and CESTMIR DVORACEK, Acta oto-laryng. **36**:412 (Dec.) 1948.

After observing 2 patients with paraplegia following meningitis, for which they had been treated with intrathecal injections of penicillin in the lumbar area, the authors were stimulated to try to find the cause of the paraplegia. They studied at autopsy the spinal cord of 19 patients that had died from purulent meningitis. Also, experimental procedures were carried out on 32 rabbits. The rabbits were treated with penicillin of a concentration of 30,000 units per cubic centimeter. They were also treated with penicillin deteriorated by time, boiling or distilled water.

The observations led them to the following conclusions: Penicillin contains a convulsive factor which is evident in injections in the lumbar area in concentrations of 5,000 units and higher, per cubic centimeter. This convulsive factor (which caused clonicospastic cramps in the rabbits) cannot be destroyed by time or by boiling of the penicillin. The cramp can be stopped by withdrawal of cerebrospinal fluid and replacement with isotonic sodium chloride solution.

On histologic study, evidence of damage was seen in slight hemorrhages in the meninges and in the spinal cord itself. Penicillin in the concentration of 1,000 units per cubic centimeter in isotonic sodium chloride solution and in 10 per cent sodium chloride solution did not cause these changes.

The late symptoms, which appear three weeks or more after the injection, are caused by the organization of the exudate which is accumulated between the posterior roots and the distal parts of the spinal cord. ROBERT LEWY, Chicago.

PURULENT MENINGITIS FOLLOWING INTRASPINAL INJECTION OF PENICILLIN. P. W. BRAESTRUP, Ugesk. f. læger **108**:1136 (Oct. 10) 1946.

Two infants, 10 and 7 months old, had otitis media. One had normal and the other probably normal spinal fluid. They were treated with 5,000 Oxford units of penicillin intrathecally dissolved in 5 cc. of saline solution. Within twenty hours both of them displayed pleocytosis of the spinal fluid (more than 10,000 leukocytes per 3 cu. mm.). No bacteria were detected. In an afebrile boy aged 2½ years the injection of 7,000 Oxford units of penicillin dissolved in 7 cc. of saline solution caused a rise in the number of cells in the spinal fluid from 9 to 2,000 per 3 cu. mm. In a girl aged 2½ months 3,000 Oxford units in 3 cc. of saline solution given intrathecally produced a rise in the cell count from 4 to 684 per 3 cu. mm. in twenty-four hours. For all the children commercial sodium penicillin was used.

WALLGREN, Stockholm, Sweden [AM. J. DIS. CHILD].

Society Transactions

AMERICAN LARYNGOLOGICAL ASSOCIATION

Frederick T. Hill, M.D., *President*

Louis H. Clerf, M.D., *Secretary*

Seventieth Annual Meeting, May 16 and 17, 1949

Abstracted by Samuel Salinger, M.D.

Factors Influencing the Growth of Lymphoid Tissue. Dr. Abraham White, Philadelphia (by invitation).

The data presented are based on animal experimentation, for the most part, from which three factors influencing the growth of lymphatic tissue are discussed. These factors are endocrine, dietary and those of stimuli arising from without. Injection of adrenotropic hormones of the anterior lobe of the pituitary gland results in involution of the thymus and the lymphatic nodes. After removal of the adrenal glands, lymphoid tissues hypertrophy. Experiments on dogs have shown that the tonsils follow the same course. Histologic examination shows shrinkage of the lymphocytes with ultimate dissolution. Secretion of the adrenotropic hormone of the pituitary gland is stimulated by various factors, such as toxins, bacteria and temperature changes. Doses of roentgen rays up to a certain point stimulate adrenocortical secretion, whereas larger doses, beyond this point, cause involution. Changes due to the sex hormones are most manifest at puberty, when involution of lymphatic tissues is commonly observed. The influence of diet is seen in cases of inanition when the decrease in caloric intake, especially of proteins, is followed by involution of lymphatic structures. Proteins are essential to the synthesis of protoplasm in the lymphoid cells. This situation also applies to deficiencies of the amino acids and pyridoxine.

DISCUSSION

Dr. Arthur W. Proetz, of St. Louis, inquired whether substances related to epinephrine, such as ephedrine and related compounds so freely employed in nose and throat practice, have any effect on the involution of lymphoid tissue.

Dr. Henry M. Goodyear, of Cincinnati, asked whether iodine has any real influence on hypertrophic lymphoid tissue. Awareness of such influence is important, in view of the widespread use of iodine in vicinities where large amounts of chlorine are used in the water supply.

Dr. A. White stated that small amounts of almost anything he has given seems to augment the pituitary cortical secretion, but it so happens that ephedrine and iodine are two of the substances he has not used experimentally. However, minute amounts of arsenic or benzene have induced involution. The influence of benzene has been known for years. It is reasonable to assume that ephedrine and iodine act in the same way, although there is no experimental evidence to support this assumption. Anesthetics such as pentobarbital sodium (nembutal®) in minute doses also act similarly. Also, it has been observed that if adrenal cortex extract is given when an animal is under anesthesia with pentobarbital sodium, the animal begins to come out of the anesthesia.

Radium Treatment of the Lymphoid Tissue of the Nasopharynx and Pharynx: A New Universal Applicator. Dr. W. Wallace Morrison, New York.

Dr. Morrison demonstrated a flexible applicator for the application of radium to the lateral pharyngeal areas when the radium is introduced through the nose. This applicator increases the area which can be reached in direct application. The indications for irradiation in the nasopharynx are recurring aerotitis and recurring acute catarrhal otitis, and irradiation is used for prevention of secondary bacterial infection, chronic suppurative otitis media and for the reduction of hypertrophied lateral bands following tonsillectomy. Contraindications are nerve deafness, otosclerosis, atrophic rhinitis and pharyngitis, congenital bursas, recent acute infection and large masses of adenoids. Danger to the operator should not be minimized. The maximum daily tolerance dose for the operator or nurse is 0.1 r per day. Adequate protection and safeguards should always be provided. Danger to the patient may arise from overdosage. The possibility of a late atrophy must also be considered.

Contact Ulcer Granuloma. Dr. Gordon B. New, Rochester, Minn.

Between 1937 and 1946, 53 patients with contact ulcer granuloma were treated at the Mayo Clinic. The condition is due to vocal abuse and is the result of forced hammering of the vocal processes against each other. Eighteen cases were caused by the pressure of intubation tubes. The resulting granuloma may be very vascular and, in some cases, may become fibrosed. The symptoms are hoarseness, cough and hemoptysis. Treatment consists of destruction of the granuloma by surgical diathermy performed under suspension laryngoscopy, with a combination of local anesthesia, the use of curare and thiopental sodium U. S. P. (pentothal sodium®) intravenously. In some cases the treatment had to be repeated after a month or two. The fibrous granulomas seemed to yield more readily than the soft friable types. Of 39 cases that were traced, the condition in 32 was found to be completely cured.

Lethal Granuloma of the Nose, Pharynx and Larynx. Dr. Henry L. Williams, Rochester, Minn.

This condition has been variously termed by different authors, and its cause is unknown. It is characterized by a slowly developing ulcer granuloma that invades the underlying tissues and the bone, which break down, causing widespread destruction, with local abscesses, sequestrums, etc., to which the patient finally succumbs from exhaustion, hemorrhage and infection. The commonest site is about the nose and mouth. About 30 to 60 cases have been recorded in the literature, and all attempts to isolate a specific organism have failed. There is some similarity to the vasculitis associated with other lesions and to periarteritis nodosa. An association with high sodium content in the blood has also been noted. Moderate success has resulted from reduction in salt intake and the administration of ammonium chloride and sugar-activated cortical hormone, together with ascorbic acid.

DISCUSSION ON PRECEDING CASES

Dr. Frederick T. Hill, of Waterville, Me., commenting on Dr. New's paper, wondered whether recurrence and the necessity for reoperation were not dependent on the type of granuloma, that is, whether it were completely sessile or pedunculated. In 1 case, in which the granuloma was bilateral, reoperation was necessary on the side where the lesion was sessile.

Dr. Paul H. Holinger, of Chicago, stated that in his experience the soft granulomas responded more readily to therapy than the fibrous types. He also noted that pain is apparently not always referred to the site of the granuloma, being frequently experienced in the opposite cord. Voice training is very important for these patients after surgical treatment. Dr. Holinger showed several colored slides typifying the lesions under discussion.

Dr. Norton Canfield, of New Haven, Conn., described 2 cases he had seen, which were similar to the ones described by Dr. Williams. Large doses of penicillin were unavailing to the second patient, who lived only six weeks after the diagnosis was made.

Dr. Fletcher D. Woodward, of Charlottesville, Va., cited a case in which a broad sessile mass occupied the entire left vocal cord. The lesion was removed endoscopically on four occasions, but still persisted. It was suspected that the underlying cartilage may have been involved, and Dr. Woodward felt that it may yet be necessary to remove the arytenoid cartilage.

Dr. Edward D. King, of Los Angeles, asked Dr. Williams whether he had obtained any results from his therapy, since Dr. Williams had not been able to finish his paper within the allotted time.

Dr. G. B. New stated that in his experience most of the cases of indeterminate type were sessile and that Dr. Holinger's colored pictures showed the lesions most beautifully.

With regard to the soft granulomas, many of which were postanesthetic, cases, the prognosis was good, since there was recovery in all, whether they were fibrosed or purely inflammatory.

Commenting on Dr. Woodward's case, Dr. New stated that he has on several occasions been compelled to remove much of the arytenoid cartilage in order to get rid of all the diseased tissue.

Dr. H. L. Williams said he had not had the opportunity to try out the suggested therapy in a case of lethal granuloma, but referred to its possibilities because of some success it has achieved in cases of lupus erythematosis and rheumatic fever.

An Internist Looks at Specialism. Dr. Joseph C. Doane, Philadelphia (by invitation).

Dr. Doane reviewed the increasing trend toward specialism, pointing out the dangers as well as the advantages. Responsibility rests heavily in the very beginning on the deans and admitting committees of medical schools and in the selection of proper candidates. The process of "cramming," so prevalent nowadays, has a bad influence on the development of the future specialist. Lack of uniformity in requirements, procedures and fees for initial and reexamination among the various specialty boards is a matter that requires coordinated study and action. Also, there is a great tendency among specialists to refer symptoms to their own particular field. A broader base of general knowledge is essential in the making of a well balanced specialist, and there is need for better group study and coordination of all factors in many cases referred to specialists. Dr. Doane aptly quoted Mosher, who once said that the strength of specialism is specialism; paradoxically, its weakness is specialism.

DISCUSSION

Dr. Burt R. Shurly, of Detroit, after fifty years in the practice of medicine, still finds the greatest joy in knowing the patient as a person. To be successful, one must be primarily a physician, rather than a specialist.

Dr. Perry G. Goldsmith, of Toronto, Canada, having practiced general medicine before becoming a specialist, agreed most emphatically with the speaker on the matter of broad general knowledge. Moreover, he is convinced that many specialists spend too little time in taking the patient's history. Since 60 per cent of the patients coming to the physician are impelled by fear or anxiety and usually have nothing the matter with them, it is extremely important that the physician be qualified to evaluate the symptoms and set the patient aright. Overtreatment and coddling of patients with minor symptoms are to be deplored.

Dr. J. C. Doane cited several examples of needless operation in cases in which the complete history was not properly evaluated. He also pointed out mistakes resulting from lack of cooperation between the dentist and the internist, and the frequently needless sacrifice of teeth.

Changes in the Nasal Mucosa Following Laryngectomy. Dr. Fred W. Dixon, Dr. Normand Hoerr and Dr. Julius McCall, of Cleveland.

Examination was made in 30 cases of laryngectomy at intervals varying from three months to eleven years after operation, and data were recorded about changes noted in the nasal mucosa. The mucosa became light purple, resembling that of the allergic nose, but was not as swollen. Few bacteria were present, sneezing was below normal and the olfactory sense somewhat diminished. The normally inactive areas of ciliary movement were speeded up, thus diminishing the emptying time. Chalk deposited in the inactive areas reached the nasopharynx, on the average, in nine and a half minutes. Temperature readings with a thermocouple revealed average temperatures of 35.2 C. (95.4 F.), as compared with 34 C. (93.2 F.) in 100 normal subjects. Biopsy specimens showed two types of tissue, vascular and fibrous; in the vascular type, the vessels were closer to the surface, the veins having very thin walls. In the fibrous type, there were bundles of collagenous fibers, with numbers of goblet cells resting on a thin basement membrane. Sparsity of round cells was noted in most of the specimens. This may account for the comparative infrequency of colds.

DISCUSSION

Dr. Julius McCall commented on the infrequency of colds in these patients. He attributes this to the fact that drying of the secretions and slowing of the ciliary movement, which in nasal respiration frequently provide the portal of entry for bacteria, did not occur because of the absence of air currents through the nose. All patients who have had a laryngectomy have moist nasal membranes, and patients who had previously suffered from sinus infections are found to be relieved.

Dr. Harold I. Lille, of Rochester, Minn., corroborated the previous speaker's statement with regard to moisture of the nasal mucosa. In 1 case in which nasal crusting had been present prior to the laryngectomy, the crusting disappeared entirely without any treatment.

Dr. Arthur W. Proetz, having examined the histologic specimens prepared by Dr. Dixon, was impressed with the normal appearance of the mucous membrane taken from patients up to 70 years. This observation goes a long way toward substantiating a feeling Dr. Proetz had had for a long time; namely, that inflammatory processes involving these tissues, however prolonged, may still be reversible, given ideal conditions, such as prevail after laryngectomy.

Dr. Andrew A. Eggston, of New York, emphasized the importance of collaboration between the clinician and the pathologist. Since the mucous membrane varies with the site, it is extremely important that the exact location from which

the specimen was removed be noted. He was much interested in the observations made in these cases and quite astounded at the minimal changes in the mucosa in the absence of nasal respiration.

Referring to the recognition of allergy of the mucosa, Dr. Eggston pointed out that plasma cells are as plentiful as eosinophils, both resulting from hyperglobulinemia.

Dr. F. W. Dixon said that all his biopsy specimens were taken from the anterior tip of the inferior turbinate bone. This location was preferred because of its accessibility and because there was less bleeding and prompter healing than had the tissue been taken from the septum.

Bony Stenosis of the Larynx: Report of a Case. Dr. Norton Canfield, New Haven, Conn.

A tracheotomy was done on a child of 2 years for the relief of dyspnea, due to an acute infection after an intubation had proved unsuccessful. Hemolytic streptococci and Pneumococcus type 16 were recovered from the larynx and Hemophilus influenzae from the trachea. The larynx filled up with papillomatous masses, which were removed. Stenosis developed, which failed to yield to repeated dilation. After nine months the larynx was still blocked, and a thyrotomy was performed. The stenosed area was reamed out and a silver tube introduced, which was later replaced with a rubber tube. The excised tissue revealed osseous fragments with well formed haversian systems and marrow spaces. Dilation was continued for about two years. The patient is now 18 years, married, has a child and is able to speak, though with a rather husky voice. Her breathing is not too good but is satisfactory for moderate activity. A record taken of the patient's voice was presented and gave an excellent reproduction of her voice and respiration.

Stenosis of the Larynx. Dr. James M. Robb, Detroit.

Dr. Robb discussed stenosis of the larynx and damage to the cartilage from intubation tubes, endotracheal tubes in anesthesia and indwelling esophageal tubes, citing typical cases. When it is imperative that such tubes be left in for some time, it is important that they be removed at intervals of a few days, cleansed and replaced, if required, by new ones. Esophageal tubes should not exceed 18 French caliber and should be made of rubber. Experiments on dogs with cellulose material, airfoam® rubber and lucite® did not show promise. It was found that young dogs did better than the older animals. Dr. Robb sought to explain by analogy the comparative rarity of stenosis in children with diphtheria, only 1 such case having been encountered in twenty years. Yet in spite of this fine record he would still prefer tracheotomy to intubation.

DISCUSSION ON PRECEDING CASES

Dr. Burt R. Shurly pointed with satisfaction to the fine record at the Harper Hospital in the handling of these cases of diphtheria. He himself had done over 500 intubations in the old days and had seen only 12 cases in which the tube had to be retained for any length of time. Stenosis was prevented by reduction in the size of the tubes, which were often coated with aluminum or silver salts. His experience with tracheotomy in cases of tuberculosis coincides with that of Dr. Robb, namely, that the results were not good. Fortunately, today with the use of streptomycin, the necessity for this procedure comes but rarely.

DR. PAUL H. HOLINGER, discussing Dr. Canfield's case, asked whether roentgenograms revealed the extent and degree of ossification. Keloid formation, more pronounced in certain persons than in others, may account for a greater tendency toward stenosis. As for indwelling esophageal tubes, Dr. Holinger believes it is not so much a question of how long the tube is in situ as it is the degree of pressure over one restricted area. Frequent examination of the larynx is necessary, especially if there is pain, and, if the tube cannot be removed, it should at least be shifted from one piriform fossa to one of the opposite side.

Dr. Robb's report was indeed remarkable in that there was only 1 case of stenosis in 1,000 intubations. When it was found that a child could not retain a tube and required one larger than was indicated for the particular age, a tracheotomy was performed in preference, an important factor in the series. The non-cough-up tube is not safe; more cases of stenosis have been seen after its use than after the use of the conventional tube. Dr. Holinger showed several slides illustrating various types of stenosis, ulceration and web formation.

DR. HAROLD G. TOBY, of Boston, stated that ulceration caused by indwelling duodenal tubes, such as the Miller-Abbott tube, could hardly be due to the caliber of the tube, which is very small. He explained the damage as due to the friction caused by the tugging on the tube, the result of peristaltic action in the small intestines.

DR. N. CANFIELD stated that roentgenograms were not taken because there was no suspicion of new bone formation. At operation, bone formation was found in the airway between the cricoid cartilage and the vocal cords themselves. The rest of the larynx looked normal, as did the cricoid itself. As to esophageal tubes, he believes the damage they cause is due to contraction of the cricopharyngeus. A new tube recently employed is collapsible. This feature has the additional advantage of preventing regurgitation.

Dr. J. M. ROBB stated that there is still room for improvement in the types of tubes introduced. His experience during the last war impressed him with the importance of the laryngologists familiarizing himself with the procedure of endotracheal anesthesia and making frequent examinations after such a procedure, as well as in cases in which esophageal feeding tubes are in use.

As a result of a poll among the members of the association, the following persons were named as having made the most outstanding contributions to laryngology:

Dr. Harris P. Mosher	Teaching and research
Dr. Thomas H. Halstead	Early work in endoscopy
Dr. George B. Wood	Anatomy, histology and histopathology of the tonsil
Dr. Chevalier Jackson	Endoscopy
Dr. Burt R. Shurly	Nutrition and diet with relation to otolaryngology
Dr. George M. Coates	Postgraduate education
Dr. Fielding O. Lewis	Surgery—malignant lesions of the larynx
Dr. Harry A. Barnes	Work on the tonsils and malignant lesions of the sinuses
Dr. Ralph Butler	Postgraduate teaching
Dr. Joseph L. Goodale	Allergy—anaphylaxis
Dr. Joseph B. Greene	Tuberculosis of the larynx
Dr. Francis R. Packard	Medical history

A number of these physicians were unable to be present at the meeting and sent letters, which were read by the secretary, in which they outlined the steps taken in their early days toward developing the special interests through which they became so well known to the profession. Dr. Mosher, Dr. Halstead, Dr. Shurly and Dr. Coates presented their outlines in person. Their presentation was followed by an evaluation and appreciation by Dr. Proetz and Dr. Lillie of the contributions of the aforementioned men. Dr. George M. Coates, of Philadelphia, who had been secretary for a number of years before being elected president, then

presented an interesting review of many sessions of the association during the period of his service, in which he recalled numerous outstanding programs of the day and the authors whose research and originality enriched the proceedings of the association.

PRESENTATION OF NEW INSTRUMENTS

DR. JOHN G. MCLAURIN, of Dallas, Tex., was reminded by Dr. Morrison's description of a new radium applicator for the pharynx of an instrument, now obsolete, which he used with success in the prevention of closure of a sphenoid aperture after operation on the sphenoid sinus. He found that the closure usually was caused by an excess of lymphoid tissue, which yielded nicely to irradiation by means of the old-time radium needle threaded into a simple cannula and left in situ for twenty to twenty-five minutes at intervals of a week.

DR. EDWIN N. BROYLES, of Baltimore, presented a new laryngoscope, whose proximal end is smaller than the distal end; this is a decided advantage when prominent teeth or a thick neck interferes with wide exposure. To increase the vision and afford a sharper view of the details of the larynx, Dr. Broyles has devised a small proximal magnifying glass, which can easily be shifted over the opening of the tube or moved out of the way as required.

DR. GABRIEL TUCKER, of Philadelphia, presented a twin-lighted laryngoscope with an attachment for oxygen insufflation, which is especially useful in the examination of the larynx and upper part of the esophagus in infants. Also, Dr. Tucker showed a new photographic outfit, made by the American Cystoscope Company, with which it is possible to obtain good pictures of the subglottic area.

Recent Advances in the Knowledge of Cancer. DR. JOSEPH C. AUB, Boston (by invitation).

Dr. Aub first spoke on diagnosis, calling attention to the most important contribution in recent years, that of the Papanicolau stain, which has proved of value in all localities where secretion from the region of the tumor is available. The test is significant because it depends on the known disposition of malignant tumors to slough off apparently viable cells, which are readily distinguishable from cells derived from normal mucosa, which are usually old cells and not always viable. Coleman showed conclusively that neoplastic cells lack the cohesiveness which characterizes normal tissue. This lack of cohesiveness accounts for the danger of implantation during operation on malignant neoplasms.

The second important advance has been in connection with radioactive isotopes. Thus far they have not been of much help in laryngology, but in the case of the pituitary and the thyroid gland radioactive iodine has already been of great service. Also, radioactive phosphorus has been useful in the diagnosis of tumors of the brain.

For diagnosis of the nasopharynx, there has as yet been little progress, so far as the use of either of the aforementioned procedures is concerned.

As for therapy, at the present, operation and irradiation are still the best there is to offer. Efforts to develop specific destructive agents against tumor cells have not been entirely successful, although the nitrogen mustards (the [β-chlorethyl] amines) have proved of value in the treatment of lymphoid tissue and some tumors of the lung. Since the nitrogen mustards act particularly on the actively dividing cells, they destroy not only the tumor cells but, at the same time, also a large number of the white cells in the blood stream, as well as the platelets; and the

destruction of these may reduce the percentages to a dangerously low level. In some cases transfusions have successfully carried the patient through the crisis to a cure.

Another new chemical having a selective action on actively dividing cells is urethane, which has been of some value in cases of myelogenous leukemia. On the other hand, folic acid has been disappointing in Dr. Aub's experience, perhaps because his work has been with adults, whereas the favorable results reported have been mostly with children.

Dr. Aub further discussed the propensity of tumor cells to divide more rapidly than normal cells and the problem of discovering ways and means to halt this rapid proliferation. A hint as to possibilities in this direction is the fact that carcinoma of the prostate gland is definitely slowed down by the use of estrogens and orchectomy. The influence of hormones is being widely explored with varying success but always with the possibility of eventual solution.

DISCUSSION

Dr. W. Wallace Morrison mentioned the use of radioactive isotopes in malignant lesions of the breast. He has had some success in detecting malignant growths of the nasopharynx with the Papanicolau stain, 12 such cases having come under his observation. This success was possible because of studies made over a period of time relating to the normal cells recoverable in the secretions of the pharynx, whereby differentiation from tumor cells was more easily determined.

Another point that has come to the fore in this work is that frequently a report of malignancy can be obtained when biopsy specimens are rubbed over a slide and the Papanicolau stain used.

Dr. Norton Canfield asked whether in view of the tendency of malignant tumors to desquamate, one should change the present method of obtaining biopsy specimens.

Dr. Andrew A. Eggston was impressed with the tendency of certain persons toward malignant growths or recurrences after removal, which, in view of the influence of hormones, seems to suggest a physiologic imbalance.

He mentioned the recent increase in malignant growths of the sinuses, as observed in the hospital with which he is associated. These, with tumors of the pharynx, present a wide variety of types, many of which are highly malignant, metastasize early and, although responding early to irradiation, are prone to recur.

While applauding the Papanicolau stain as a remarkable advance, Dr. Eggston believes that the clinician should not rely on it completely, since it places too great a responsibility on the pathologist. A new procedure, which has been of value at times, is to rub a piece of absorbable gelatin sponge (gelfoam®) over the suspected area, then drop it into formaldehyde solution (formalin®), section and stain, as for a biopsy. Frequently, bits of tumor tissue are found which present typical characteristics of malignancy.

Dr. Louis H. Clerf, of Philadelphia, had found the Papanicolau stain of value in the diagnosis of endobronchial lesions and suggested the possibility of its being used in suspected malignant growth of the sinus, where secretions may contain numbers of malignant cells.

Dr. J. C. Aub expressed a fear of taking specimens for biopsy in many cases because of the possibility of thereby spreading and implanting tumor cells. Certainly, the procedure should be carried out with extreme care.

He spoke of the virus question, which was brought up by one of the discussers. While the effect of the virus in animals has been proved with experiments, its relation to carcinoma in the human being is still conjectural. It seems logical, however, to assume that viruses may stimulate the mechanism whereby cells are activated to rapid propagation. The relation of nucleoprotein to tumor cells is intriguing, because it has been shown that cells seem to derive the energy for propagation from this biological. This has been shown in experiments on the liver, where amino acids can activate a small rest of tissue to overfunctionate after the greater part of the organ has been removed. Tumor cells have shown a greater aptitude in this direction, which again brings up the great question of why normal cells will, in certain persons, tend the same way.

Plastic Repair of Saddle Nose. Dr. Edward D. King, Los Angeles.

Dr. King has used cancellous bone in 20 cases of saddle nose with good results. The bone is obtained from the iliac crest and is inserted either through a tip or a vestibular incision. The nasal bones are rasped before the implant is introduced, a procedure which facilitates union between the bones and the implant. The implanted bone soon is invaded by new blood vessels, and new bone is formed to replace the portions initially absorbed.

DISCUSSION

Dr. Samuel Salinger, of Chicago, said that the greatest objection to bone implants is the fact that the resulting nose is too rigid and incapable of the mobility which is normally inherent by reason of the exposed position of the organ. His own preference is for autogenous cartilage and, when this is not available, for cadaver cartilage of proper consistency. He recalled his experiences with all these materials and said that in some instances neither cartilage nor bone will suffice. This is particularly true in post-traumatic and postinfection cases in which there is extreme retraction of the tip of the nose and columella, owing to dense scarring. For this type of case he has had good results with ivory, properly prepared and made up in two interlocking pieces to fill the nasal dorsum and support the tip and columella. Several cases of this type have been under observation for more than twenty years and no apparent deleterious effects were noted.

Dr. Fred Z. Havens, of Rochester, Minn., also commented on the rigidity resulting from the use of bone grafts. His own preference is for autogenous rib cartilage, which is put into a double boiler before use in order to prevent curling.

Dr. E. D. King admitted that the nose does feel rigid after a bone implant, but believes that as long as it looks well and patients are satisfied, the means are justified.

Hemilaryngectomy: A Method of Maintaining a Permanent and Ample Airway. Dr. Henry M. Goodyear, Cincinnati.

Dr. Goodyear suggested that in cases in which the carcinoma was limited to one half of the larynx but too extensive for a simple thyrotomy, removal of half of the larynx would leave the patient a normal airway. The technic shown involves removal of the pretracheal muscles, resection of the ala of the thyroid and arytenoid cartilage. Mucosa from the pharynx is drawn in to line the raw area, and a solid acrylic tube 7.5 by 1.3 cm. is inserted, with perforations enabling

it to be fitted over the tracheal tube. This tube is worn for several weeks and the tracheal tube retained up to from ten to twelve weeks. Laryngeal speech and breathing are gradually reestablished.

DISCUSSION

Dr. Louis H. Clerf expressed interest in the procedure, although he had never seen it done. He believed it a pity to sacrifice half a larynx if it could be saved.

Dr. Gordon B. New inquired about the grade of tumor involved. He would consider the procedure risky if the tumor were a highly malignant one, because the manipulation involved would be likely to scatter tumor cells into raw areas. He said he would be inclined to destroy the tumor with surgical diathermy, which he considered safer than the cold knife. As to resultant contraction, Dr. New had had good results from the implantation of a skin graft, having observed several cases up to three years with the graft plainly visible.

Dr. H. M. Goodyear admitted that the procedure was new and had not been under observation in a sufficient number of cases or for a sufficiently long period to afford a complete evaluation. However, he thought the procedure should be explored because of the possibility of rehabilitating certain patients who come midway between the group who are ideal candidates for laryngofissure and the group that must have a laryngectomy. He agreed with Dr. New on the value of electrocoagulation, which he uses in part of the operation. As for the skin graft in cases of stenosis, he had seen the skin ultimately replaced with mucous membrane, which is contrary to Dr. New's experience. Dr. Goodyear's case may have been an exceptional one.

Book Reviews

Hearing Tests and Hearing Instruments. By Leland A. Watson and Thomas Tolan, M.D. Price, $7. Pp. 600. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore, 1949.

This 600 page book is a pioneer in and major contribution to the field of audiology. It is written in a clear and concise manner and explains the numerous complicated instruments and procedures commonly used to test hearing. The chapters on the engineering aspects of the audiometer and on hearing aids are lengthy and complicated for the otologist but are invaluable as reference material. The chapters on the interpretations of audiometric data and social aspects of audiometry are brilliantly written. In a mature manner and with careful editing, the book presents practically all the accumulated knowledge that is worth knowing in this field. Everyone interested in the ear should have this book.

The 47 page bibliography is in itself a fine contribution. The authors have avoided discussing old theories and useless procedures once so prevalent in this specialty. Instead, they present not only accurate and proved technics for testing hearing, but also the reasons for their use and their limitations. The graphs and illustrations are clear and descriptive. The authors have presented a comprehensive and completely authoritative book on the hearing tests and hearing instruments.

Clinical Audiology. By Maurice Saltzman, M.D. With an introduction by George M. Coates, M.D., and Matthew S. Ersner, M.D. Price, $5. Pp. 195. Grune & Stratton, Inc., 381 4th Ave., New York, 1949.

This comprehensive book fills a need in covering the ever increasing mass of literature which has been appearing in recent years on the subject of deafness. Strangely enough, more than half of this literature has come from men who are not otologists. Physicists have contributed facts on the physics of sound; physiologists have dealt with the physiology of hearing; psychologists have taken up central perception, and educators have written on the importance of hearing in relation to the learning of language, the acquisition of speech and the development of education. All phases of the subject are here touched on, necessarily briefly, in a volume of about 200 pages.

In spite of the author's wide familiarity with the subject, the approach to and explanation of those writings outside the field of clinical otology are difficult to read for one not schooled in the language of physicists, acoustic engineers and psychologists. In addition, difficulties arise from the fact that much of the work on the physiology and psychology of hearing is in a controversial field. It is not yet known exactly how sound is heard and interpreted. Theories have not led one very far as yet.

There is much, however, that is practical in the book, particularly in the analyses of the different types of deafness, the use of speech-hearing testing, the selection of hearing aids, malingering testing, the rating of disability and the

rehabilitation of the deaf. The practicing otologist may wish for a more particular exposition of individual hearing tests and more information on what the tests reveal.

All in all, the book is most stimulating to careful thinking on a most important subject in otology.

Public School Audiometry, Principles and Methods. By Loraine A. Dahl. Price, not given. Pp. 290. The Interstate Printers, 19-27 N. Jackson St., Danville, Ill., 1949.

This illustrated textbook on the testing of hearing of school children is thorough and practical. It will serve an excellent purpose in training and in orienting lay workers in this field. As a "field manual" it covers instrumental manipulations and the handling of public and professional relations, a knowledge of which these lay technicians need. As Dr. Livile stresses in his preface, the book fits in well with the hearing conservation program about to be initiated by the American Academy of Ophthalmology and Otolaryngology. "It will serve to bring about more uniform and more reliable audiometric procedures."

The book is written in a most clear and concise style, and the material is well organized and consecutive. Otologists will profit greatly by familiarizing themselves with the technics of speech and frequency testing as set forth here. The format of the book suffers from the absence of a table of contents, and the reproduction of the photographs might well be improved on.

Allergy in Relation to Otolaryngology. By French K. Hansel, M.D. Price, $2.50. Pp. 75. The Bruce Publishing Company, 2462 University Ave., St. Paul, 1949. Published by the American College of Allergists, Inc.

This short book is a panel discussion opened by Dr. Hansel on the subject of allergy in otolaryngology. In his talk, the author discusses the major points with regard to diagnosis and treatment of allergy and how important it is to have excellent cooperation between allergists and otolaryngologists. Many interesting and significant problems are introduced by Dr. Hansel and his discussers. The relation of allergy to aviation medicine and bronchology are brought out, in addition to the important problem of surgery of the paranasal sinuses. The panel discussion is interesting and stimulating to read.

Theory of Hearing. By Ernest Glen Wever. Price, $6. Pp. 484. John Wiley & Sons, Inc., 440 4th Ave., New York, 1949.

Written by one of the pioneer investigators in the field of hearing, this book very lucidly reviews the development of the auditory theory to the present concept. There are three main divisions. The first section presents an excellent historical evaluation of the earlier theories of hearing and an explanation of how and why they developed. The second section deals with the two most prominent theories, that on resonance and on frequency, each of which is thoroughly and critically reviewed. In the third section a new theory, the Volley theory, is developed on the background of the previous work. Evidence for the validity of this theory is presented, and clinical phenomena are explained.

The book contains a wealth of interesting material with excellent criticism by the author. The new Volley theory combines the numerous virtues of the two previous major theories and, in many instances, obviates many of their shortcomings. The book should be read by everyone interested in the physiology and pathology of hearing.

Audiology: The Science of Hearing, a Developing Professional Specialty. By Norton Canfield, M.D. With a foreword by Gunnar Holmgren. Price, $1.75. Pp. 45. Charles C Thomas, Publisher, 220 E. Monroe St., Springfield, Ill., 1949.

Nothing quite as comprehensive, concise and direct has appeared as this 45 page octavo brief on the current trends in handling the problems of deafness. The book is an outline of the "Audiology Center" of the future, with all aspects of deafness covered. There are chapters on the roles of the acoustic physicist, psychologist, testing technician, sociologist, speech therapist and otologist. It will indeed be a welcome day when such groups of persons can cooperate toward the accomplishment of relief of the many-sided handicap.

An excellent bibliography completes the book. The format and printing are unusually attractive.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Fourth International Congress of Otolaryngology

President: Dr. V. E. Negus, London, England.
General Secretaries: Dr. F. C. W. Capps and Dr. W. A. Mill, 45 Lincoln's Inn Fields, London, W. C. 2.
Place: London. Time: July 18-23, 1949.

Second Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Prof. Justo Alonso.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Montevideo. Time: January 1950.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. William H. Johnston, 1515 State St., Santa Barbara, Calif.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit 26, Mich.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles 14.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Leroy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-26, 1950.

American Laryngological Association

President: Dr. Ralph A. Fenton, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Hotel Mark Hopkins, San Francisco. Time: May 23-24, 1950.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-27, 1950.

SECTIONS:

Eastern.—Chairman: Dr. Robert L. Moorhead, 125 Remsen St., Brooklyn, N. Y.
Southern.—Chairman: Dr. William D. Stimson, 899 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. J. Marion Sutherland, 3001 W. Grand Blvd., Detroit.
Western.—Chairman: Dr. J. B. Naftzger, 6777 Hollywood Blvd., Hollywood, Calif.

American Otological Society

President: Dr. Philip E. Meltzer, 20 Charlesgate, W., Boston.
Secretary: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 3, N Y.
Place: Hotel Mark Hopkins, San Francisco. Time: May 21-22, 1950.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Samuel F. Kelley, 47 E. 61st St., New York 21.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. George E. Shambaugh Jr., 55 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Joseph W. Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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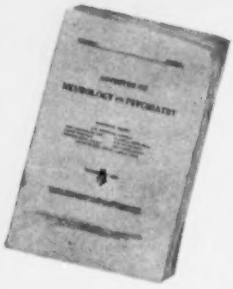

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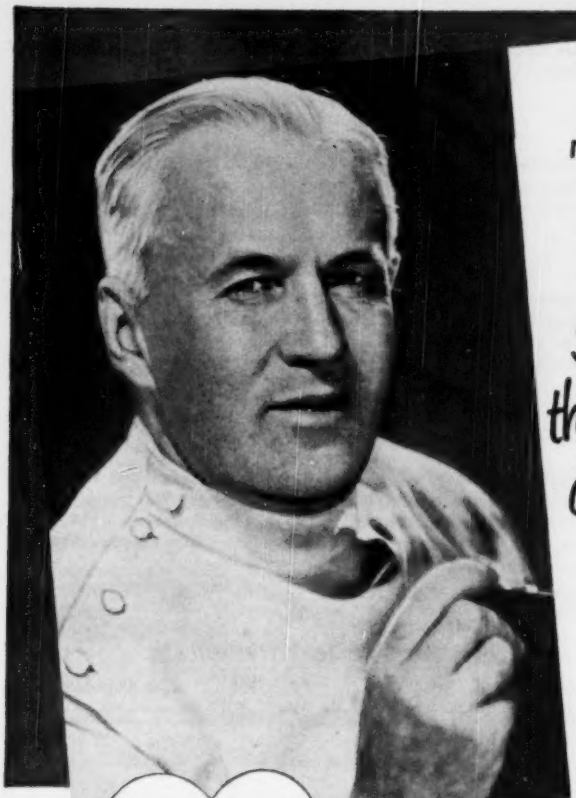

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